Psychopathology notes

Clinical Psychology (Amity University)

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NEURODEVELOPMENTAL DISORDERS

1. Intellectual Disability

Intellectual disability, formerly known as mental retardation, can be caused by a range of environmental and genetic factors that lead to a combination of cognitive and social impairments. The American Association on Intellectual and Developmental Disability (AAIDD) defines intellectual disability as a disability characterized by significant limitations in both intellectual functioning (reasoning, learning, and problem solving) and in adaptive behavior (conceptual, social, and practical skills) that emerges before the age of 18 years. In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), various levels of severity of intellectual disability are determined on the basis of adaptive functioning, not on IQ scores. Making a determination of severity level of intellectual disability, according to DSM-5, includes assessment of functioning in a conceptual domain (e.g., academic skills), a social domain (e.g., relationships), and a practical domain (e.g., personal hygiene).

Council for Exceptional Children (CEC) and the National Association for Retarded Citizens (NARC)

DEGREES OF SEVERITY OF INTELLECTUAL DISABILITY:

Mild intellectual disability represents approximately 85 percent of persons with intellectual disability. Children with mild intellectual disability often are not identified until the first or second grade, when academic demands increase. By late adolescence, they often acquire academic skills at approximately a sixth-grade level. Specific causes for the intellectual disability are often unidentified in this group. Many adults with mild intellectual disability can live independently with appropriate support and raise their own families. IQ for this level of adaptive function may typically range from 50 to 70.

Moderate intellectual disability represents about 10 percent of persons with intellectual disability. Most children with moderate intellectual disability acquire language and can communicate adequately during early childhood. They are challenged academically and often are not able to achieve above a second to third grade level. During adolescence, socialization difficulties often set these persons apart, and a great deal of social and vocational support is beneficial. As adults, individuals with moderate intellectual disability may be able to perform semiskilled work under appropriate supervision. IQ for this level of adaptive function may typically range from 35 to 50.

Severe intellectual disability represents about 4 percent of individuals with intellectual disability. They may be able to develop communication skills in childhood and often can learn to count as well as recognize words that are critical to functioning. In this group, the cause for the intellectual disability is more likely to be identified than in milder forms of intellectual disability. In adulthood, persons with severe intellectual disability may adapt well to supervised living situations, such as

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group homes, and may be able to perform work-related tasks under supervision. IQ in individuals with this level of adaptive function may typically range from 20 to 35.

Profound intellectual disability constitutes approximately 1 to 2 percent of individuals with intellectual disability. Most individuals with profound intellectual disability have identifiable causes for their condition. Children with profound intellectual disability may be taught some self-care skills and learn to communicate their needs given the appropriate training. IQ in individuals with this level of adaptive function may typically be less than 20.

EPIDEMIOLOGY:

The majority of population-based prevalence estimates for intellectual disability in developing countries range from 10 to 15 per 1,000 children. The prevalence of intellectual disability at any one time is estimated to range from 1 to 3 percent of the population in Western societies. The highest incidence of intellectual disability is reported in school-age children, with the peak at ages 10 to 14 years. Intellectual disability is about 1.5 times more common among males than females.

ETIOLOGY:

Genetic causes include chromosomal and inherited conditions; developmental and environmental factors include prenatal exposure to infections and toxins; and environmental or acquired factors include prenatal trauma (e.g., prematurity) and sociocultural factors. Among chromosomal disorders, Down syndrome and fragile X syndrome are the most common disorders that usually produce at least moderate intellectual disability. A prototype of a metabolic disorder associated with intellectual disability is phenylketonuria (PKU). Deprivation of nutrition, nurturance, and social stimulation can potentially contribute to the development of at least mild forms of intellectual disability.

Genetic Etiological Factors in Intellectual Disability:

Single-Gene Causes-

One of the most well-known single gene causes of intellectually disability is found in the FMR1 gene whose mutations cause fragile X syndrome. It is the most common and first X-linked gene to be identified as a direct cause of intellectual disability. Abnormalities in autosomal chromosomes are frequently associated with intellectual disability.

Visible and Submicroscopic Chromosomal Causes of Intellectual Disability-

Trisomy 21 (Down syndrome) is a prototype of a cytogenetically visible abnormality that accounts for about two-thirds of the 15 percent of intellectual disability attributable to visible abnormal cytogenetics. Other microscopically visible chromosomal abnormalities associated with intellectual disability include deletions, translocations, and supernumerary marker chromosomes. Typically, microscopic chromosome analysis is able to identify abnormalities of 5 to 10 million base pairs or greater.

Environmental and Sociocultural Factors:

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Mild intellectual disability has been associated with significant deprivation of nutrition and nurturance. Children who have endured these conditions are at risk for a host of psychiatric disorders including mood disorders, posttraumatic stress disorder, and attentional and anxiety disorders. Prenatal environment compromised by poor medical care and poor maternal nutrition may be contributing factors in the development of mild intellectual disability. Teenage pregnancies are at risk for mild intellectual disability in the baby due to the increased risk of obstetrical complications, prematurity, and low birth weight. Poor postnatal medical care, malnutrition, exposure to toxic substances such as lead, and potential physical trauma are additional risk factors for mild intellectual disabilities. Child neglect and inadequate caretaking may deprive an infant of both physical and emotional nurturances, leading to failure to thrive syndromes.

DIAGNOSIS:

The diagnosis of intellectual disability can be made after the history is obtained, using information from a standardized intellectual assessment, and a standardized measure of adaptive function indicating that a child is significantly below the expected level in both areas. The severity of the intellectual disability will be determined on the basis of the level of adaptive function. A history and psychiatric interview are useful in obtaining a longitudinal picture of the child's development and functioning. Examination of physical signs, neurological abnormalities, and in some cases, laboratory tests can be used to ascertain the cause and prognosis.

CLINICAL FEATURES:

Mild intellectual disability may not be recognized or diagnosed in a child until school challenges the child's social and communication skills. Cognitive deficits include poor ability to abstract and egocentric thinking, both of which become more easily evident as a child reaches middle childhood. Children with milder intellectual disabilities may function academically at the high elementary level and may acquire vocational skills sufficient to support themselves in some cases; however, social assimilation may be problematic. Communication deficits, poor self-esteem, and dependence may further contribute to a relative lack of social spontaneity.

Moderate levels of intellectual disability are significantly more likely to be observed at a younger age, since communication skills develop more slowly and social isolation may ensue in the elementary school years. Academic achievement is usually limited to the middle-elementary level. Children with moderate intellectual deficits benefit from individual attention focused on the development of self-help skills. However, these children are aware of their deficits and often feel alienated from their peers and frustrated by their limitations. They continue to require a relatively high level of supervision but can become competent at occupational tasks in supportive settings.

Severe intellectual disability is typically obvious in the preschool years; affected children have minimal speech and impaired motor development. Some language development may occur in the school-age years. By adolescence, if language has not improved significantly, poor, nonverbal forms of communication may have evolved. Behavioral approaches are useful means to promote some self-care, although those with severe intellectual disability generally need extensive supervision.

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Children with profound intellectual disability require constant supervision and are severely limited in both communication and motor skills. By adulthood, some speech development may be present, and simple self-help skills may be acquired. Clinical features frequently observed in populations with intellectual disability either in isolation or as part of a mental disorder, include hyperactivity, low frustration tolerance, aggression, affective instability, repetitive and stereotypic motor behaviors, and self-injurious behaviors. Self-injurious behaviors occur more frequently and with greater intensity in more severe intellectual disability.

COURSE AND PROGNOSIS:

Although the underlying intellectual impairment does not improve, in most cases of intellectual disability, level of adaptation increases with age and can be influenced positively by an enriched and supportive environment. In general, persons with mild and moderate mental intellectual disabilities have the most flexibility in adapting to various environmental conditions. Comorbid psychiatric disorders negatively impact overall prognosis. When psychiatric disorders are superimposed on intellectual disability, standard treatments for the comorbid mental disorders are often beneficial; however, less robust responses and increased vulnerability to side effects of psychopharmacologic agents are often the case.

DIFFERENTIAL DIAGNOSIS:

Sensory disabilities, especially deafness and blindness, can be mistaken for intellectual disability when a lack of awareness of the sensory deficit leads to inappropriate testing. Expressive and receptive speech disorders may give the impression of intellectual disability in a child of average intelligence, and cerebral palsy may be mistaken for intellectual disability. Chronic, debilitating medical diseases may depress and delay a child's functioning and achievement, despite normal intelligence. Seizure disorders, especially those that are poorly controlled, may contribute to persisting intellectual disability. Specific organic syndromes leading to isolated handicaps such as failure to read (alexia), failure to write (agraphia), or failure to communicate (aphasia), may occur in a child of normal and even superior intelligence. Children with learning disorders (which can coexist with intellectual disability) experience a delay or failure of development in a specific area, such as reading or mathematics, but they develop normally in other areas. In contrast, children with intellectual disability show general delays in most areas of development. Intellectual disability and autism spectrum disorder (ASD) often coexist; 70 to 75 percent of those with ASD have an IQ below 70. In addition, epidemiologic data indicate that ASD occurs in approximately 19.8% of persons with intellectual disability. Children with ASD have relatively more severe impairment in social relatedness and language than other children with the same level of intellectual disability. A child younger than the age of 18 years with significant adaptive functional impairment, with an IQ less than 70, who also meets diagnostic criteria for dementia, will receive both a diagnosis of dementia and intellectual disability. However, a child whose IQ drops below 70 after the age of 18 years with newly acquired cognitive impairment will receive only the diagnosis of dementia.

TREATMENT:

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Interventions for children and adolescents with intellectual disability are based on an assessment of social, educational, psychiatric, and environmental needs. Intellectual disability is associated with a variety of comorbid psychiatric disorders that often require specific treatment, in addition to psychosocial support. Of course, when preventive measures are available, the optimal approach includes primary, secondary, and tertiary interventions.

Primary Prevention-

Primary prevention comprises actions taken to eliminate or reduce the conditions that lead to development of intellectual disability, as well as associated disorders. For example, screening babies for PKU, and administrating a low phenylalanine diet when PKU is present, significantly alters the emergence of intellectual disability in those affected children. Additional primary prevention steps include education of the general public about strategies to prevent intellectual disability, such as abstinence from alcohol during pregnancy; continuing efforts of health professionals to ensure and upgrade public health policies; and legislation to provide optimal maternal and child health care. Family and genetic counseling helps reduce the incidence of intellectual disability in a family with a history of a genetic disorder.

Secondary and Tertiary Prevention-

Prompt attention to medical and psychiatric complications of intellectual disability can diminish their course (secondary prevention) and minimize the sequelae or consequent disabilities (tertiary prevention). Hereditary metabolic and endocrine disorders, such as PKU and hypothyroidism, can be treated effectively in an early stage by dietary control or hormone replacement therapy.

2. Autism Spectrum Disorder

Autism spectrum disorder, previously known as the pervasive developmental disorders, is a phenotypically heterogeneous group of neurodevelopmental syndromes, with polygenic heritability, characterized by a wide range of impairments in social communication and restricted and repetitive behaviors. Autistic disorder was characterized by impairments in three domains: social communication, restricted and repetitive behaviors, and aberrant language development and usage. Recent clinical consensus has shifted the conceptualization of autism spectrum disorder toward a continuum model in which heterogeneity of symptoms is recognized as inherent in the disorder, and core diagnostic impairments are collapsed into two domains: deficits in social communication, and restricted and repetitive behaviors. Aberrant language development and usage is no longer considered a core feature of autism spectrum disorder. Autism spectrum disorder is typically evident during the second year of life, and in severe cases, a lack of developmentally appropriate interest in social interactions may be noted even in the first year. Children with autism spectrum disorder often exhibit idiosyncratic intense interest in a narrow range of activities, resist change, and typically, do not respond to their social environment in accordance with their peers. According to the DSM-5, diagnostic criteria for autism spectrum disorder include deficits in social communication and

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restricted interests, which present in the early developmental period, however, when subtle, may not be identified until several years later.

Repetitive behaviors such as hand-flapping, toe-walking, and odd play were also identified with autism spectrum disorder disorders at a younger age.

EPIDEMIOLOGY:

Prevalence-

Autism spectrum disorders have been increasingly diagnosed over the last two decades, with the current prevalence estimated at approximately 1 percent in the United States. Autistic disorder, based on DSM-IV-TR criteria, is believed to occur at a rate of about 8 cases per 10,000 children (0.08 percent). By definition, the onset of autism spectrum disorder is in the early developmental period; however, some cases are not recognized until the child is much older. Because of this delay between onset and diagnosis, the prevalence rates increase with age in young children.

Sex Distribution-

Autism spectrum disorder is diagnosed four times more often in boys than in girls. In clinical samples, girls with autism spectrum disorder more often exhibit intellectual disability than boys. One potential explanation for this is that girls with autism spectrum disorder without intellectual disability may be less likely to be identified, referred clinically, and diagnosed.

ETIOLOGY:

Genetic Factors-

Although up to 15 percent of cases of autism spectrum disorder appear to be associated with a known genetic mutation, in most cases, its expression is dependent on multiple genes. The concordance rate of autistic disorder in two large twin studies was 36 percent in monozygotic pairs versus 0 percent in dizygotic pairs in one study and about 96 percent in monozygotic pairs versus about 27 percent in dizygotic pairs in the second study. High rates of cognitive impairments, in the non-autistic twin in monozygotic twins with perinatal complications, suggest that contributions of perinatal environmental factors interact with genetic vulnerability differentially in autism spectrum disorder. Genetic studies have identified two biological systems that are influenced in autism spectrum disorder: the consistent finding of elevated platelet serotonin (5-HT), and the mTOR, that is, mammalian target of rapamycin–linked synaptic plasticity mechanisms, which appear to be disrupted in autism spectrum disorder. Researchers who screened the DNA of more than 150 pairs of siblings with autism spectrum disorder found evidence of two regions on chromosomes 2 and 7 containing genes that may contribute to autism spectrum disorder. Additional genes hypothesized to be involved in autism spectrum disorder were found on chromosomes 16 and 17.

Biomarkers-

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Elevated serotonin in whole blood, almost exclusively in the platelets. Platelets acquire 5-HT through the process of SERT (serotonin transporter), known to be hereditary, as they pass through the intestinal circulation. The genes that mediate SERT (SLC64A), and the 5-HT receptor 5-HT 2A gene (HTR2A) are known to be more heritable than autism spectrum disorder, and encode the same protein in the platelets and in the brain. Because 5-HT is known to be involved in brain development, it is possible that the changes in 5-HT regulation may lead to alterations in neuronal migration and growth in the brain.

Psychosocial Theories-

Studies comparing parents of children with autism spectrum disorder with parents of normal children have shown no significant differences in child-rearing skills. Kanner's early speculations that parental emotional factors might be implicated as contributing to the development of autism spectrum disorder have been clearly refuted.

Core Symptoms of Autism Spectrum Disorder:

- Persistent Deficits in Social Communication and Interaction
- Restricted, Repetitive Patterns of Behavior, Interests, and Activities
- Associated Physical Characteristics

DIFFERENTIAL DIAGNOSIS:

Disorders to consider in the differential diagnosis of autism spectrum disorder include social (pragmatic) communication disorder, the newly described DSM-5 communication disorder; schizophrenia with childhood onset; congenital deafness or severe hearing disorder; and psychosocial deprivation. It is also difficult to make the diagnosis of autism spectrum disorder because of its potentially overlapping symptoms with childhood schizophrenia, intellectual disability syndromes with behavioral symptoms, and language disorders.

COURSE AND PROGNOSIS:

Autism spectrum disorder is typically a lifelong, albeit heterogeneous, disorder with a highly variable severity and prognosis. Children with autism spectrum disorder and IQs above 70 with average adaptive skills, who develop communicative language by ages 5 to 7 years, have the best prognoses. A longitudinal study comparing symptoms in children with high-IQ autism spectrum disorder at the age of 5 years, with their symptoms at age 13 through young adulthood, found that a small proportion no longer met criteria for autism spectrum disorder.

TREATMENT:

The goals of treatment for children with autism spectrum disorder are to target core behaviors to improve social interactions, communication, broaden strategies to integrate into schools, develop meaningful peer relationships, and increase long-term skills in independent living. Psychosocial treatment interventions aim to help children with autism spectrum disorder to develop skills in social

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conventions, increase socially acceptable and pro-social behavior with peers, and to decrease odd behavioral symptoms. In many cases, language and academic remediation are also required. In addition, treatment goals generally include reduction of irritable and disruptive behaviors that may emerge in school and at home and may exacerbate during transitions. In addition, parents of children with autism spectrum disorder often benefit from psycho-education, support, and counseling in order to optimize their relationships and effectiveness with their children. Comprehensive treatment for autism spectrum disorder including intensive behavioral programs, parent training and participation, and academic/educational interventions have provided the most promising results.

International Classification of Diseases, Tenth Edition (ICD-10):

The classification system used in International Classification of Diseases, Tenth Revision (ICD-10) is not congruent with the revisions made in DSM-5 about autistic disorders. ICD-10 still includes separate designations for Rett syndrome, Childhood Disintegrative Disorder, Asperger's Disorder, and Pervasive Development Disorder Not Otherwise Specified. The reader should be aware, however, that according to DSM-5, each is subsumed under the rubric of Autism Spectrum Disorder and should so be diagnosed.

3. Attention Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) is a neuropsychiatric condition affecting preschoolers, children, adolescents, and adults around the world, characterized by a pattern of diminished sustained attention, and increased impulsivity or hyperactivity. Although multiple regions of the brain and several neurotransmitters have been implicated in the emergence of symptoms, dopamine continues to be a focus of investigation regarding ADHD symptoms. The prefrontal cortex of the brain has been implicated because of its high utilization of dopamine and its reciprocal connections with other brain regions involved in attention, inhibition, decision-making, response inhibition, working memory, and vigilance. ADHD affects up to 5 to 8 percent of school-aged children, with 60 to 85 percent of those diagnosed as children continuing to meet criteria for the disorder in adolescence, and up to 60 percent continuing to be symptomatic into adulthood. Children, adolescents, and adults with ADHD often have significant impairment in academic functioning as well as in social and interpersonal situations. ADHD is frequently associated with comorbid disorders including learning disorders, anxiety disorders, mood disorders, and disruptive behavior disorders.

The Fifth Edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM5) has made several changes to the diagnostic criteria of ADHD in youth and in adults. Whereas in the past, ADHD symptoms had to be present by age 7 years, in DSM-5, "several inattentive or hyperactive-impulsive symptoms" must be present by age 12 years. Previously, there were two subtypes: Inattentive and Hyperactive/Impulsive type. In DSM-5, however, subtypes have been replaced by the following three specifiers, which essentially denote the same groups: (1) combined presentation, (2) predominantly inattentive presentation, and (3) predominantly hyperactive/impulsive presentation. Additional changes in DSM-5 include permitting a comorbid

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ADHD and autism spectrum diagnosis to be made. Finally, in DSM-5, for adolescents 17 years and older and for adults, only five symptoms, rather than six symptoms of either inattention or hyperactivity and impulsivity are required. In addition, to reflect the developmental differences in ADHD across the life span, examples of symptoms have been added to the DSM-5 criteria for ADHD. To confirm a diagnosis of ADHD, impairment from inattention and/or hyperactivity and impulsivity must be present in at least two settings and interfere with developmentally appropriate social or academic functioning.

EPIDEMIOLOGY:

Rates of ADHD have been reported to be 7 to 8 percent in prepubertal elementary school children. Epidemiologic studies suggest that ADHD occurs in about 5 percent of youth including children and adolescents, and about 2.5 percent of adults. The rate of ADHD in parents and siblings of children with ADHD is 2 to 8 times greater than in the general population. ADHD is more prevalent in boys than in girls, with the ratio ranging from 2:1 to as high as 9:1. First-degree biological relatives (e.g., siblings of probands with ADHD) are at high risk for developing ADHD. The parents of children with ADHD show an increased incidence of substance use disorders. Symptoms of ADHD are often present by age 3 years, but unless they are very severe, the diagnosis is frequently not made until the child is in kindergarten, or elementary school, when teacher information is available comparing the index child peers of the same age.

ETIOLOGY:

Data suggest that the etiology of ADHD is largely genetic, with a heritability of approximately 75 percent. Most children with ADHD have no evidence of gross structural damage in the central nervous system (CNS).

Genetic Factors-

Cook and colleagues have found an association of the dopamine transporter gene (DAT1) with ADHD, although data from other research groups have not confirmed that result. Family studies and populationbased studies have found an association between the dopamine 4 receptor seven-repeat allele gene (DRD4) gene and ADHD. Most molecular research on ADHD has focused on genes that influence the metabolism or action of dopamine.

Neurochemical Factors-

Many neurotransmitters are postulated to be associated with ADHD symptoms; however, dopamine is a major focus of clinical investigation, and the prefrontal cortex has been implicated based on its role in attention and regulation of impulse control. Animal studies have shown that other brain regions such as locus ceruleus, which consists predominantly of noradrenergic neurons, also play a major role in attention. Dysfunction in peripheral epinephrine, which causes the hormone to accumulate peripherally, may potentially feed back to the central system and “reset” the locus ceruleus to a lower level.

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Neurophysiological Factors-

EEG studies in ADHD children and adolescents over the last several decades have found evidence of increased theta activity, especially in the frontal regions. Further studies of youth with ADHD have provided data showing elevated beta activity in their electroencephalography (EEG). Show increased mood lability and temper tantrums.

Neuroanatomical Aspects-

A review of magnetic resonance imaging (MRI), positron emission tomography (PET), and single photon emission computerized tomography (SPECT) suggests that populations of children with ADHD show evidence of both decreased volume and decreased activity in prefrontal regions, anterior cingulated, globus pallidus, caudate, thalamus, and cerebellum.

Developmental Factors-

Higher rates of ADHD are present in children who were born prematurely and whose mothers were observed to have maternal infection during pregnancy. Perinatal insult to the brain during early infancy caused by infection, inflammation, and trauma may, in some cases, be contributing factors in the emergence of ADHD symptoms.

Psychosocial Factors-

Severe chronic abuse, maltreatment, and neglect are associated with certain behavioral symptoms that overlap with ADHD including poor attention and poor impulse control. Predisposing factors may include the child's temperament and genetic–familial factors.

DIAGNOSIS:

The diagnosis of ADHD requires persistent, impairing symptoms of either hyperactivity/impulsivity or inattention in at least two different settings. For example, most children with ADHD have symptoms in school and at home.

Distinguishing features of ADHD are short attention span and high levels of distractibility for chronological age and developmental level. In school, children with ADHD often exhibit difficulties following instructions and require increased individualized attention from teachers. At home, children with ADHD frequently have difficulty complying with their parents' directions and may need to be asked multiple times to complete relatively simple tasks. Children with ADHD typically act impulsively, are emotionally labile, explosive, lack focus, and are irritable. Exhibit comorbid conduct disorder than those children with inattentive ADHD. Specific learning disorders in the areas of reading, arithmetic, language, and writing occur frequently in association with ADHD. Global developmental assessment must be considered to rule out other sources of inattention.

A child with ADHD may exhibit distractibility and perseveration and signs of visual-perceptual, auditory-perceptual, or languagebased learning disorders. A neurological examination may reveal

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visual, motor, perceptual, or auditory discriminatory immaturity or impairments without overt signs of visual or auditory disorders. Children with ADHD often have problems with motor coordination and difficulty copying age-appropriate figures, rapid alternating movements, right–left discrimination, ambidexterity, reflex asymmetries, and a variety of subtle nonfocal neurological signs (soft signs).

CLINICAL FEATURES:

ADHD can have its onset in infancy, although it is rarely recognized until a child is at least toddler age. More commonly, infants with ADHD are active in the crib, sleep little, and cry a great deal. In school, children with ADHD may attack a test rapidly, but may answer only the first two questions. They may be unable to wait to be called on in school and may respond before everyone else. At home, they cannot be put off for even a minute. Impulsiveness and an inability to delay gratification are characteristic. Children with ADHD are often susceptible to accidents. The most cited characteristics of children with ADHD, in order of frequency, are hyperactivity, attention deficit (short attention span, distractibility, perseveration, failure to finish tasks, inattention, poor concentration), impulsivity (action before thought, abrupt shifts in activity, lack of organization, jumping up in class), memory and thinking deficits, specific learning disabilities, and speech and hearing deficits. Associated features often include perceptual motor impairment, emotional lability, and developmental coordination disorder. A significant percent of children with ADHD show behavioral symptoms of aggression and defiance. School difficulties, both learning and behavioral, commonly exist with ADHD. Comorbid communication disorders or learning disorders that hamper the acquisition, retention, and display of knowledge complicate the course of ADHD.

DIFFERENTIAL DIAGNOSIS:

A temperamental constellation of high activity level and short attention span, in the normal range for the child's age, and without impairment, should be ruled out.

Anxiety in a child needs to be evaluated. Anxiety can accompany ADHD as a symptom or comorbid disorder, and anxiety can manifest with overactivity and easy distractibility. Mania and ADHD share many core features, such as excessive verbalization, motoric hyperactivity, and high levels of distractibility. Although mania and ADHD can coexist, children with bipolar I disorder exhibit more waxing and waning of symptoms than those with ADHD. Oppositional defiant disorder, or conduct disorder and ADHD may coexist, and when that occurs, both disorders are diagnosed. Specific learning disorders of various kinds must also be distinguished from ADHD; a child may be unable to read or do mathematics because of a learning disorder, rather than because of inattention. ADHD often coexists with one or more learning problems, including deficits in reading, mathematics or written expression.

COURSE AND PROGNOSIS:

The course of ADHD is variable. Symptoms have been shown to persist into adolescence in 60 to 85 percent of cases, and into adult life in approximately 60 percent of cases. The remaining 40 percent

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of cases may remit at puberty, or in early adulthood. In some cases, the hyperactivity may disappear, but the decreased attention span and impulsecontrol problems persist. Overactivity is usually the first symptom to remit, and distractibility is the last. ADHD does not usually remit during middle childhood. Children with ADHD whose symptoms persist into adolescence are at higher risk for developing conduct disorder.

Most children with ADHD have some social difficulties. Socially dysfunctional children with ADHD have significantly higher rates of comorbid psychiatric disorders, and experience more problems with behavior in school as well as with peers and family members. Overall, the outcome of ADHD in childhood seems to be related to the degree of persistent comorbid psychopathology, especially conduct disorder, social disability, and chaotic family factors. Optimal outcomes may be promoted by ameliorating children's social functioning, diminishing aggression, and improving family situations as early as possible.

TREATMENT:

In medically healthy youth, however, excellent safety records are documented for short- and sustained-release preparations of methylphenidate (Ritalin, Ritalin-SR, Concerta, Metadate CD, Metadate ER), dextroamphetamine (Dexedrine, Dexedrine spansules, Vyvanse), and dextroamphetamine and amphetamine salt combinations (Adderall, Adderall XR). Newer preparations of methylphenidate, include Methylin, a chewable form of methylphenidate; Daytrana, a methylphenidate patch; and dexmethylphenidate, the denantiomer (Focalin), and its longer acting form Focalin XR. These newer preparations aim to maximize the target effects and minimize the adverse effects in individuals with ADHD who obtain partial response from methylphenidate or whose dose was limited by side effects. Vyvanse (lisdexamfetamine dimesylate) is a pro-drug of dextroamphetamine, which requires intestinal metabolism in order to reach its active form

Current strategies favor once a day sustained-release stimulant preparations for their convenience and diminished rebound side effects. Advantages of the sustained-release preparations for children are that one dose in the morning will sustain the effects all day, and the child is no longer required to interrupt his or her school day, as well as the physiologic advantage that the medication is sustained at an approximately even level in the body throughout the day so that periods of rebound and irritability are avoided.

Nonstimulant medications approved by the FDA in the treatment of ADHD include atomoxetine (Strattera), a norepinephrine uptake inhibitor. A-agonists including clonidine (Catapres) and guanfacine (Tenex) have also been found to be effective in treating ADHD. The FDA has recently approved the extended-release forms of clonidine (Kapvay) and the extended release form of guanfacine (Intuniv) for the treatment of ADHD in children 6 years and older. Antidepressants, such as bupropion (Wellbutrin, Wellbutrin SR), have been used with variable success in the treatment of ADHD.

4. Specific Learning Disorder

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Specific learning disorder in youth is a neurodevelopmental disorder produced by the interactions of heritable and environmental factors that influence the brain's ability to efficiently perceive or process verbal and nonverbal information. It is characterized by persistent difficulty learning academic skills in reading, written expression, or mathematics, beginning in early childhood, that is inconsistent with the overall intellectual ability of a child.

The American Psychiatric Association's Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM5), combines the DSM-IV diagnoses of reading disorder, mathematics disorder, and disorder of written expression and learning disorder not otherwise specified into a single diagnosis: Specific learning disorder. Learning deficits in reading, written expression, and mathematics in the DSM-5 are designated using specifiers. DSM-5 notes that the term dyslexia is an equivalent term describing a pattern of learning difficulties, including deficits in accurate or fluent word recognition, poor decoding, and poor spelling skills. Dyscalculia is noted to be an alternative term referring to a pattern of deficits related to learning arithmetic facts, processing numerical information, and performing accurate calculations.

Specific learning disorder of all types affects approximately 10 percent of youth.

A recent epidemiologic study found four profiles including (1) weak reading, (2) weak language, (3) weak math, or (4) combined weak math and reading, accounting for 70 percent of children with specific learning impairments. Specific learning disorder is associated with an increased risk of comorbid disorders, including attention-deficit/hyperactivity disorder (ADHD), communication disorders, conduct disorders, and depressive disorders. Moderate to high heritability is believed to contribute to specific learning disorder, and furthermore, it appears that many cognitive traits are polygenic. In addition, there is pleiotropy, that is, the same genes may affect skills necessary for diverse learning tasks. Factors such as perinatal injury and specific neurological conditions may contribute to the development of specific learning disorder. Conditions such as lead poisoning, fetal alcohol syndrome, and in utero drug exposure are also associated with increased rates of specific learning disorder.

SPECIFIC LEARNING DISORDER WITH IMPAIRMENT IN READING-DYSLEXIA

Reading impairment is present in up to 75 percent of children and adolescents with specific learning disorder. Students who have learning problems in other academic areas most commonly experience difficulties with reading as well. Reading impairment is characterized by difficulty in recognizing words, slow and inaccurate reading, poor comprehension, and difficulties with spelling. Reading impairment is often comorbid with other disorders in children, particularly, ADHD.

Dyslexia was used extensively for many years to describe a reading disability syndrome that often included speech and language deficits and right–left confusion. Reading impairment is frequently accompanied by disabilities in other academic skills, and the term dyslexia remains as an alternate term for a pattern of reading and spelling difficulties.

EPIDEMIOLOGY:

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An estimated 4 to 8 percent of youth in the United States have been identified with dyslexia, encompassing a variety of reading, spelling, and comprehension deficits. Three to four times as many boys as girls are reported to have reading impairments in clinically referred samples. In epidemiological samples, however, rates of reading impairments are much closer among boys and girls. Boys with reading impairment are referred for psychiatric evaluation more often than girls due to comorbid ADHD and disruptive behavior problems. No clear gender differential is seen among adults who report reading difficulties.

ETIOLOGY:

Because reading impairment typically includes a language deficit, the left brain has been hypothesized to be the anatomical site of this dysfunction. Several studies using magnetic resonance imaging (MRI) studies have suggested that the planum temporale in the left brain shows less asymmetry than the same site in the right brain in children with both language disorders and specific learning disorder. Positron emission tomographic (PET) studies have led some researchers to conclude that left temporal blood flow patterns during language tasks differ between children with and without learning disorders. Cell analysis studies suggest that in reading impaired individuals, the visual magnocellular system (which normally contains large cells) contains more disorganized and smaller cell bodies than expected. Several studies have suggested that phonological awareness (i.e., the ability to decode sounds and sound out words) is linked to chromosome 6. Furthermore, the ability to identify single words has been linked to chromosome 15. Impairment in reading and spelling has now been linked to susceptibility loci on multiple chromosomes, including chromosomes 1, 2, 3, 6, 15, and 18.

DIAGNOSIS:

Reading impairment is diagnosed when a child's reading achievement is significantly below that expected of a child of the same age. Characteristic diagnostic features include difficulty recalling, evoking, and sequencing printed letters and words; processing sophisticated grammatical constructions; and making inferences.

CLINICAL FEATURES:

Children with reading disabilities are usually identified by the age of 7 years (second grade). Children can sometimes compensate for reading disorder in the early elementary grades by the use of memory and inference, particularly in children with high intelligence. In such instances, the disorder may not be apparent until age 9 (fourth grade) or later. Children with reading impairment make many errors in their oral reading. The errors are characterized by omissions, additions, and distortions of words. Such children have difficulty in distinguishing between printed letter characters and sizes, especially those that differ only in spatial orientation and length of line. The child's reading speed is slow, often with minimal comprehension. Most children with reading disability have an age-appropriate ability to copy from a written or printed text, but nearly all spell poorly.

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Associated problems include language difficulties: discrimination and difficulty in sequencing words properly. A child with reading disorders may start a word either in the middle or at the end of a printed or written sentence. Most children with reading disorder dislike and avoid reading and writing. Their anxiety is heightened when they are confronted with demands that involve printed language. Older children tend to be angry and depressed and exhibit poor self-esteem.

COURSE AND PROGNOSIS:

Children with reading disability may gain knowledge of printed language during their first 2 years in grade school, without remedial assistance. By the end of the first grade, many children with reading problems, in fact, have learned how to read a few words; however, by the third grade, keeping up with classmates is exceedingly difficult without remedial educational intervention. When remediation is instituted early, in milder cases, it may not be necessary after the first or second grade. In severe cases and depending on the pattern of deficits and strengths, remediation may be continued into the middle and high school years.

DIFFERENTIAL DIAGNOSIS:

Reading deficits are often accompanied by comorbid disorders, such as language disorder, disability in written expression, and ADHD. Deficits in expressive language and speech discrimination along with reading disorder may lead to a comorbid diagnosis of language disorder. Reading impairment must be differentiated from intellectual disability syndromes in which reading, along with most other skills, are below the achievement expected for a child's chronological age. Intellectual testing helps to differentiate global deficits from more specific reading difficulties. Hearing and visual impairments should be ruled out with screening tests.

TREATMENT:

Remediation strategies for children with reading impairments focus on direct instruction that leads a child's attention to the connections between speech sounds and spelling. Children and adolescents with reading difficulties are entitled to an individual education program (IEP) provided by the public school system. High schoolers with persisting reading difficulties may have greater benefit from individualized reading remediation. Reading instruction programs such as the Orton Gillingham and Direct Instructional System for Teaching and Remediation (DISTAR) approaches begin by concentrating on individual letters and sounds, advance to the mastery of simple phonetic units, and then blend these units into words and sentences. Thus, if children are taught to cope with graphemes, they will learn to read. Other reading remediation programs, such as the Merrill program, and the Science Research Associates, Inc. (SRA) Basic Reading Program, begin by introducing whole words first and then teach children how to break them down and recognize the sounds of the syllables and the individual letters in the word. Another approach teaches children with reading disorders to recognize whole words through the use of visual aids and bypasses the sounding-out process. One such program is called the Bridge Reading Program. The Fernald method uses a multisensory approach that combines teaching whole words with a tracing technique so that the child has kinesthetic stimulation while learning to read the words.

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SPECIFIC LEARNING DISORDER WITH IMPAIRMENT IN MATHEMATICS- DYSCALCULIA

Children with mathematics difficulties have difficulty learning and remembering numerals, cannot remember basic facts about numbers, and are slow and inaccurate in computation. Poor achievement in four groups of skills have been identified in mathematics disorder: linguistic skills (those related to understanding mathematical terms and converting written problems into mathematical symbols), perceptual skills (the ability to recognize and understand symbols and order clusters of numbers), mathematical skills (basic addition, subtraction, multiplication, division, and following sequencing of basic operations), and attentional skills (copying figures correctly and observing operational symbols correctly). Mathematics deficits can, however, occur in isolation or in conjunction with language and reading impairments. According to the DSM-5, the diagnosis of specific learning disorder with impairment in mathematics consists of deficits in arithmetic counting and calculations, has difficulty remembering mathematics facts, and may count on fingers instead. Additional deficits include difficulty with mathematic concepts and reasoning, leading to difficulties in applying procedures to solve quantitative problems.

EPIDEMIOLOGY:

Mathematics disability alone is estimated to occur in about 1 percent of school-age children, that is about one of every five children with specific learning disorder. Epidemiological studies have indicated that up to 6 percent of school-age children have some difficulty with mathematics, and prevalence estimates of 3.5 to 6.5 percent have been reported for impairing forms of dyscalculia. Although specific learning disorder overall occurs two to three times more often in males, mathematics deficits may be relatively more frequent in girls than reading deficits. Many studies of learning disorders in children have grouped reading, writing, and mathematics disability together, which makes it more difficult to ascertain the precise prevalence of mathematics disability.

ETIOLOGY:

Mathematics deficiency, as with other areas of specific learning disorder, has a significant genetic contribution. High rates of comorbidity with reading deficits have been reported in the range of 17 percent up to 60 percent. One theory proposed a neurological deficit in the right cerebral hemisphere, particularly in the occipital lobe areas. These regions are responsible for processing visual–spatial stimuli that, in turn, are responsible for mathematical skills. This theory, however, has received little support in subsequent neuropsychiatric studies. Causes of deficits in mathematics are believed to be multifactorial, including genetic, maturational, cognitive, emotional, educational, and socioeconomic factors. Prematurity and very low birth weight are also a risk factor for specific learning disorder, including mathematics. Compared with reading abilities, arithmetic abilities seem to depend more on the amount and quality of instruction.

DIAGNOSIS:

The diagnosis of specific learning disorder in mathematics is made when a child's skill in mathematical reasoning, or calculation, remain significantly below what is expected for that child's

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age, for a period of at least 6 months, even when remedial interventions have been administered. Many different skills contribute to mathematics proficiency. These include linguistic skills, conceptual skills, and computational skills. Linguistic skills involve being able to understand mathematical terms, understand word problems, and translate them into the proper mathematical process. Conceptual skills involve recognition of mathematical symbols and being able to use mathematical signs correctly. Computational skills include the ability to line up numbers correctly and to follow the “rules” of the mathematical operation.

CLINICAL FEATURES:

Common features of mathematics deficit include difficulty learning number names, remembering the signs for addition and subtraction, learning multiplication tables, translating word problems into computations, and performing calculations at the expected pace. Most children with mathematics deficits can be detected during the second and third grades in elementary school. Some investigators have classified mathematics deficiencies into the following categories: difficulty learning to count meaningfully; difficulty mastering cardinal and ordinal systems; difficulty performing arithmetic operations; and difficulty envisioning clusters of objects as groups. Children with mathematics difficulty have trouble associating auditory and visual symbols, understanding the conservation of quantity, remembering sequences of arithmetic steps, and choosing principles for problem-solving activities. Mathematics difficulty, in fact, often coexists with other disorders affecting reading, expressive writing, coordination, and language. Spelling problems, deficits in memory or attention, and emotional or behavioral problems may be present.

COURSE AND PROGNOSIS:

A child with a specific learning disorder in mathematics can usually be identified by the age of 8 years (third grade). In some children, the disorder is apparent as early as 6 years (first grade); in others, it may not be apparent until age 10 (fifth grade) or later. Children with a moderate mathematics disorder who do not receive intervention may have complications, including continuing academic difficulties, shame, poor self-concept, frustration, and depression. These complications can lead to reluctance to attend school, and demoralization about academic success.

DIFFERENTIAL DIAGNOSIS:

Mathematics deficits must be differentiated from global causes of impaired functioning such as intellectual disability. Arithmetic difficulties in intellectual disability are accompanied by similar impairments in overall intellectual functioning. Inadequate schooling can affect a child’s arithmetic performance on a standardized arithmetic test. Conduct disorder or ADHD can occur comorbidly with specific learning disorder in mathematics and, in these cases, both diagnoses should be made.

TREATMENT:

Mathematics difficulties for children are best remediated with early interventions that lead to improved skills in basic computation. The presence of specific learning disorder in reading along with

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mathematics difficulties can impede progress; however, children are quite responsive to remediation in early grade school. Children with indications of mathematics disorder as early as in kindergarten require help in understanding which digit in a pair is larger, counting abilities, identification of numbers, and remembering sequences of numbers. Flash cards, workbooks, and computer games can be a viable part of this treatment. One study indicated that mathematics instruction is most helpful when the focus is on problem-solving activities, including word problems, rather than only computation. Project MATH, a multimedia self-instructional or group-instructional in-service training program, has been successful for some children with mathematics disorder. Computer programs can be helpful and can increase compliance with remediation efforts. Social skills deficits can contribute to a child's hesitation in asking for help, so a child identified with a mathematics disorder may benefit from gaining positive problemsolving skills in the social arena as well as in mathematics.

SPECIFIC LEARNING DISORDER WITH IMPAIMRENT IN WRITTEN EXPRESSION-DYSGRAPHIA

EPIDEMIOLOGY:

The prevalence of specific learning disorder with impairment in written expression has been reported to occur in the range of 5 to –15 percent of school-age children. Over time, specific learning disorder remits in many youth, leading to a persistent rate of specific learning disorder of 4 percent in adults. The gender ratio in writing deficits is two to three to one in boys compared with girls. Impaired written expression often occurs along with deficits in reading, but not always.

ETIOLOGY:

Causes of writing disability are believed to be similar to those of reading disorder, that is, underlying deficits in using the components of language related to letter sounds. Genetic factors are a significant factor in the development of writing disability. Writing difficulties often accompany language disorder, leading an affected child to have trouble with understanding grammatical rules, finding words, and expressing ideas clearly. According to one hypothesis, impairment in written expression may result from combined effects of language disorder, and reading disorder. Hereditary predisposition to writing impairment is supported by the finding that most youth with impaired written expression have first-degree relatives with similar difficulties Children with limited attention spans and high levels of distractibility may find writing an arduous task.

DIAGNOSIS:

The DSM-5 diagnosis of specific learning disorder with impairment in written expression is based on a child's poor ability to use punctuation and grammar accurately in sentences, inability to organize paragraphs, or to clearly articulate ideas in writing. Poor performance on composing written text may also include poor handwriting and impaired ability to spell and to place words sequentially in coherent sentences, compared to others of the same age. In addition to spelling mistakes, youth with impaired written expression make grammatical mistakes, such as using incorrect tenses, forgetting words in sentences, and placing words in the wrong order. Punctuation may be incorrect,

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and the child may have poor ability to remember which words begin with capital letters. Additional symptoms of impaired written expression include the formation of letters that are not legible, inverted letters, and mixtures of capital and lowercase letters in a given word. Other features of writing disorders include poor organization of written stories, which lack critical elements such as “where,” “when,” and “who” or clear expression of the plot.

CLINICAL FEATURES:

Youth with impairments in written expression struggle early in grade school with spelling words and expressing their thoughts according to age-appropriate grammatical norms. Their spoken and written sentences contain an unusually large number of grammatical errors and poor paragraph organization. Affected children commonly make simple grammatical errors, even when writing a short sentence. For example, despite constant reminders, affected youth frequently fail to capitalize the first letter of the first word in a sentence, and fail to end the sentence with a period. Typical features of impaired written expression include spelling errors, grammatical errors, punctuation errors, poor paragraph organization, and poor handwriting. In higher grades in school, affected youth’s written sentences become more conspicuously primitive, odd, and inaccurate compared to what is expected of students at their grade level. For youth with impaired written expression, word choices are often erroneous and inappropriate, paragraphs are disorganized and not in proper sequence, and spelling accuracy becomes increasingly difficult as their vocabulary becomes larger and more abstract. Associated features of writing impairments may include reluctance to go to school, refusal to do assigned written homework, and concurrent academic difficulties in other areas. Many children with impaired written expression understandably become frustrated and angry, and harbor feelings of shame and inadequacy regarding poor academic achievement. In some cases, depressive disorders can result from a growing sense of isolation, estrangement, and despair. Young adults with impaired written expression who do not receive remedial intervention continue to have writing skills deficits and a persistent sense of incompetence and inferiority.

COURSE AND PROGNOSIS:

Specific learning disorder with impairment in writing, reading, and mathematics often coexist, and additional language disorder may be present as well. A child with all of the above disabilities will likely be diagnosed with language disorder first and impaired written expression last. In severe cases, an impaired written expression is apparent by age 7 (second grade); in less severe cases, the disorder may not be apparent until age 10 (fifth grade) or later. Youth with mild and moderate impairment in written expression fare well if they receive timely remedial education early in grade school. Severely impaired written expression requires continual, extensive remedial treatment through the late part of high school and even into college. The prognosis depends on the severity of the disorder, the age or grade when the remedial intervention is started, the length and continuity of treatment, and presence or absence of associated or secondary emotional or behavioral problems.

DIFFERENTIAL DIAGNOSIS:

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It is important to determine whether disorders such as ADHD or major depression are interfering with a child's focus and thereby preventing the production of adequate writing in the absence of a specific writing impairment. If true, treatment for the other disorder should improve a child's writing performance. Commonly comorbid disorders with writing disability are language disorder, mathematics disorder, developmental coordination disorder, disruptive behavior disorders, and ADHD.

TREATMENT:

Remedial treatment for writing disability includes direct practice in spelling and sentence writing as well as a review of grammatical rules. Intensive and continuous administration of individually tailored, one-on-one expressive and creative writing therapy appears to effect favorable outcome. Teachers in some special schools devote as much as 2 hours a day to such writing instruction. The effectiveness of a writing intervention depends largely on an optimal relationship between the child and the writing specialist. Success or failure in sustaining the patient's motivation greatly affects the treatment's long-term efficacy. Associated secondary emotional and behavioral problems should be given prompt attention, with appropriate psychiatric treatment and parental counseling.

5. Persistent (Chronic) Motor or Vocal Tic Disorder

Chronic motor or vocal tic disorder is defined as the presence of either motor tics or vocal tics, but not both. Tics may wax and wane but must have persisted for more than 1 year since the first tic onset to meet the diagnosis for persistent (chronic) motor or vocal tic disorder. According to the Fifth Edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria, this disorder must have its onset before the age of 18 years. Chronic motor or vocal tic disorder cannot be diagnosed if the criteria for Tourette's disorder have ever been met.

EPIDEMIOLOGY:

The rate of chronic motor or vocal tic disorder has been estimated to be 100 to 1,000 times greater than that of Tourette's disorder in school-age children. School-age boys are at highest risk. Although the disorder was once believed to be rare, current estimates of the prevalence of chronic motor or vocal tic disorder range from 1 to 2 percent.

ETIOLOGY:

Chronic motor or vocal tic disorder as well as Tourette's disorder tend to aggregate in the same families. Twin studies have found a high concordance for either Tourette's disorder or chronic motor tics in monozygotic twins. This finding supports the importance of hereditary factors in the transmission of tic disorders.

DIAGNOSIS AND CLINICAL FEATURES:

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The onset of chronic motor or vocal tic disorder typically occurs in early childhood. Chronic vocal tics are considerably rarer than chronic motor tics. Chronic vocal tics, in the absence of motor tics, are typically less conspicuous than the vocal tics in Tourette's disorder. The vocal tics are usually not loud or intense and are not primarily produced by the vocal cords; they consist of grunts or other noises caused by thoracic, abdominal, or diaphragmatic contractions.

DIFFERENTIAL DIAGNOSIS:

Chronic motor tics must be differentiated from a variety of other motor movements, including choreiform movements, myoclonus, restless legs syndrome, akathisia, and dystonias. Involuntary vocal utterances can occur in certain neurological disorders, such as Huntington's disease and Parkinson's disease.

COURSE AND PROGNOSIS:

Children whose tics emerge between the ages of 6 and 8 years seem to have the best outcomes. Symptoms often last for 4 to 6 years and remit in early adolescence. Children whose tics involve the limbs or trunk may have less prompt remission than those with only facial tics.

TREATMENT:

The treatment of chronic motor or vocal tic disorder depends on several factors including the severity and frequency of the tics; the patient's subjective distress; the effects of the tics on school or work, job performance, and socialization; and the presence of any other concomitant mental disorder. Psychotherapy may be indicated to minimize the secondary social difficulties caused by severe tics. Behavioral techniques, particularly habit reversal treatments, are effective in treating chronic motor or vocal tic disorder. When severe, tics may be reduced through the use of atypical antipsychotics such as risperidone. If not effective, typical antipsychotics such as pimozide or haloperidol may be helpful. Behavioral interventions are the first line of treatment.

MOOD DISORDERS

1. Bipolar I Disorder

The DSM-5 criteria for a bipolar I disorder (Table 8.1-6) requires the presence of a distinct period of abnormal mood lasting at least 1 week and includes separate bipolar I disorder diagnoses for a single manic episode and a recurrent episode based on the symptoms of the most recent episode.

The designation bipolar I disorder is synonymous with what was formerly known as bipolar disorder—a syndrome in which a complete set of mania symptoms occurs during the course of the disorder. The diagnostic criteria for bipolar II disorder is characterized by depressive episodes and hypomanic episodes during the course of the disorder, but the episodes of manic-like symptoms do not quite meet the diagnostic criteria for a full manic syndrome.

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Manic episodes clearly precipitated by antidepressant treatment (e.g., pharmacotherapy, electroconvulsive therapy [ECT]) do not indicate bipolar I disorder.

Bipolar I Disorder, Single Manic Episode-

According to DSM-5, patients must be experiencing their first manic episode to meet the diagnostic criteria for bipolar I disorder, single manic episode. This requirement rests on the fact that patients who are having their first episode of bipolar I disorder depression cannot be distinguished from patients with major depressive disorder.

Bipolar I Disorder, Recurrent-

The issues about defining the end of an episode of depression also apply to defining the end of an episode of mania. Manic episodes are considered distinct when they are separated by at least 2 months without significant symptoms of mania or hypomania.

CLINICAL FEATURES:

DIFFERENTIAL DIAGNOSIS:

When a patient with bipolar I disorder has a depressive episode, the differential diagnosis is the same as that for a patient being considered for a diagnosis of major depressive disorder. When a patient is manic, however, the differential diagnosis includes bipolar I disorder, bipolar II disorder, cyclothymic disorder, mood disorder caused by a general medical condition, and substance-induced mood disorder. For manic symptoms, borderline, narcissistic, histrionic, and antisocial personality disorders need special consideration.

COURSE AND PROGNOSIS:

Course-

The natural history of bipolar I disorder is such that it is often useful to make a graph of a patient's disorder and to keep it up to date as treatment progresses. Although cyclothymic disorder is sometimes diagnosed retrospectively in patients with bipolar I disorder, no identified personality traits are specifically associated with bipolar I disorder. Bipolar I disorder most often starts with depression (75 percent of the time in women, 67 percent in men) and is a recurring disorder. Most patients experience both depressive and manic episodes, although 10 to 20 percent experience only manic episodes. The manic episodes typically have a rapid onset (hours or days) but may evolve over a few weeks. An untreated manic episode lasts about 3 months; therefore, clinicians should not discontinue giving drugs before that time. Of persons who have a single manic episode, 90 percent are likely to have another. As the disorder progresses, the time between episodes often decreases. After about five episodes, however, the interepisode interval often stabilizes at 6 to 9 months. Of persons with bipolar disorder, 5 to 15 percent have four or more episodes per year and can be classified as rapid cyclers.

BIPOLAR I DISORDER IN CHILDREN AND OLDER PERSONS-

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Bipolar I disorder can affect both the very young and older persons. The incidence of bipolar I disorder in children and adolescents is about 1 percent, and the onset can be as early as age 8 years. Common misdiagnoses are schizophrenia and oppositional defiant disorder. Bipolar I disorder with such an early onset is associated with a poor prognosis. Manic symptoms are common in older persons, although the range of causes is broad and includes nonpsychiatric medical conditions, dementia, and delirium, as well as bipolar I disorder. The onset of true bipolar I disorder in older persons is relatively uncommon.

Prognosis-

Patients with bipolar I disorder have a poorer prognosis than do patients with major depressive disorder. About 40 to 50 percent of patients with bipolar I disorder may have a second manic episode within 2 years of the first episode. Although lithium prophylaxis improves the course and prognosis of bipolar I disorder, probably only 50 to 60 percent of patients achieve significant control of their symptoms with lithium. One 4-year follow-up study of patients with bipolar I disorder found that a premorbid poor occupational status, alcohol dependence, psychotic features, depressive features, interepisode depressive features, and male gender were all factors that contributed a poor prognosis. Short duration of manic episodes, advanced age of onset, few suicidal thoughts, and few coexisting psychiatric or medical problems predict a better outcome. About 7 percent of patients with bipolar I disorder do not have a recurrence of symptoms; 45 percent have more than one episode, and 40 percent have a chronic disorder. Patients may have from two to 30 manic episodes, although the mean number is about nine. About 40 percent of all patients have more than ten episodes. On longterm follow-up, 15 percent of all patients with bipolar I disorder are well, 45 percent are well but have multiple relapses, 30 percent are in partial remission, and 10 percent are chronically ill. One third of all patients with bipolar I disorder have chronic symptoms and evidence of significant social decline.

TREATMENT:

2. Bipolar II Disorder

The diagnostic criteria for bipolar II disorder specify the particular severity, frequency, and duration of the hypomanic symptoms. The diagnostic criteria for a hypomanic episode are listed together with the criteria for bipolar II disorder (also in Table 8.1-6). The criteria have been established to decrease overdiagnosis of hypomanic episodes and the incorrect classification of patients with major depressive disorder as patients with bipolar II disorder. Clinically, psychiatrists may find it difficult to distinguish euthymia from hypomania in a patient who has been chronically depressed for many months or years. As with bipolar I disorder, antidepressant-induced hypomanic episodes are not diagnostic of bipolar II disorder.

CLINICAL FEATURES:

The clinical features of bipolar II disorder are those of major depressive disorder combined with those of a hypomanic episode. Although the data are limited, a few studies indicate that bipolar II disorder is

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associated with more marital disruption and with onset at an earlier age than bipolar I disorder. Evidence also indicates that patients with bipolar II disorder are at greater risk of both attempting and completing suicide than patients with bipolar I disorder and major depressive disorder.

DIFFERENTIAL DIAGNOSIS:

The differential diagnosis of patients being evaluated for a mood disorder should include the other mood disorders, psychotic disorders, and borderline disorder. The differentiation between major depressive disorder and bipolar I disorder, on one hand, and bipolar II disorder, on the other hand, rests on the clinical evaluation of the manialike episodes. Clinicians should not mistake euthymia in a chronically depressed patient for a hypomanic or manic episode. Patients with borderline personality disorder often have a severely disrupted life, similar to that of patients with bipolar II disorder, because of the multiple episodes of significant mood disorder symptoms.

COURSE AND PROGNOSIS:

The course and prognosis of bipolar II disorder indicate that the diagnosis is stable because there is a high likelihood that patients with bipolar II disorder will have the same diagnosis up to 5 years later. Bipolar II disorder is a chronic disease that warrants long-term treatment strategies.

TREATMENT:

The pharmacological treatment of bipolar disorders is divided into both acute and maintenance phases. Bipolar treatment, however, also involves the formulation of different strategies for the patient who is experiencing mania or hypomania or depression. Lithium and its augmentation by antidepressants, antipsychotics, and benzodiazepines has been the major approach to the illness, but three anticonvulsant mood stabilizers—carbamazepine (Tegretol), valproate (Depakene), and lamotrigine (Lamictal)—have been added more recently, as well as a series of atypical antipsychotics, most of which are approved for the treatment of acute mania, one also for monotherapy of acute depression, and three for prophylactic treatment. Each of these medications is associated with a unique side effect and safety profile, and no one drug is predictably effective for all patients. Often, it is necessary to try different medications before an optimal treatment is found.

3. DYSTHYMIA

The most typical features of dysthymia, also known as persistent depressive disorder, is the presence of a depressed mood that lasts most of the day and is present almost continuously. There are associated feelings of inadequacy, guilt, irritability, and anger; withdrawal from society; loss of interest; and inactivity and lack of productivity. Dysthymia is distinguished from major depressive disorder by the fact that patients complain that they have always been depressed. Thus, most cases are of early onset, beginning in childhood or adolescence and certainly occurring by the time patients reach their 20s. Although dysthymia can occur as a secondary complication of other psychiatric disorders, the core concept of dysthymia refers to a subaffective or subclinical depressive disorder with (1) low-grade

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chronicity for at least 2 years; (2) insidious onset, with origin often in childhood or adolescence; and (3) a persistent or intermittent course. The family history of patients with dysthymia is typically replete with both depressive and bipolar disorders, which is one of the more robust findings supporting its link to primary mood disorder.

EPIDEMIOLOGY:

Dysthymia is common among the general population and affects 5 to 6 percent of all persons. It is seen among patients in general psychiatric clinics, where it affects between half and one-third of all patients. No gender differences are seen for incidence rates. The disorder is more common in women younger than 64 years of age than in men of any age and is more common among unmarried and young persons and in those with low incomes. Dysthymia frequently coexists with other mental disorders, particularly major depressive disorder, and in persons with major depressive disorder, there is less likelihood of full remission between episodes. The patients may also have coexisting anxiety disorders (especially panic disorder), substance abuse, and borderline personality disorder. The disorder is more common among those with first-degree relatives with major depressive disorder. Patients with dysthymia are likely to be taking a wide range of psychiatric medications, including antidepressants, antimanic agents such as lithium (Eskalith) and carbamazepine (Tegretol), and sedative-hypnotics.

ETIOLOGY:

Biological Factor-

The biological basis for the symptoms of dysthymia and major depressive disorder are similar, but the biological bases for the underlying pathophysiology in the two disorders differ.

SLEEP STUDIES-

Decreased rapid eye movement (REM) latency and increased REM density are two state markers of depression in major depressive disorder that also occur in a significant proportion of patients with dysthymia.

NEUROENDOCRINE STUDIES-

The two most studied neuroendocrine axes in major depressive disorder and dysthymia are the adrenal axis and the thyroid axis, which have been tested by using the dexamethasone-suppression test (DST) and the thyrotropin-releasing hormone (TRH)-stimulation test, respectively. Although the results of studies are not absolutely consistent, most indicate that patients with dysthymia are less likely to have abnormal results on a DST than are patients with major depressive disorder.

Psychosocial Factors-

Psychodynamic theories about the development of dysthymia posit that the disorder results from personality and ego development and culminates in difficulty adapting to adolescence and young adulthood. Karl Abraham, for example, thought that the conflicts of depression center on oral- and anal-

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sadistic traits. Anal traits include excessive orderliness, guilt, and concern for others; they are postulated to be a defense against preoccupation with anal matter and with disorganization, hostility, and self-preoccupation. A major defense mechanism used is reaction formation. Low self-esteem, anhedonia, and introversion are often associated with the depressive character.

DIAGNOSIS AND CLINICAL FEATURES:

The DSM-5 diagnosis criteria for dysthymia (Table 8.2-1) stipulate the presence of a depressed mood most of the time for at least 2 years (or 1 year for children and adolescents). To meet the diagnostic criteria, a patient should not have symptoms that are better accounted for as major depressive disorder and should never have had a manic or hypomanic episode. DSM-5 allows clinicians to specify whether the onset was early (before age 21 years) or late (age 21 years or older). DSM-5 also allows specification of atypical features in dysthymia.

The profile of dysthymia overlaps with that of major depressive disorder but differs from it in that symptoms tend to outnumber signs (more subjective than objective depression). This means that disturbances in appetite and libido are uncharacteristic, and psychomotor agitation or retardation is not observed. Subtle endogenous features are observed, however, including inertia, lethargy, and anhedonia that are characteristically worse in the morning. Because patients presenting clinically often fluctuate in and out of a major depression, the core DSM-5 criteria for dysthymia tend to emphasize vegetative dysfunction; however, cognitive symptoms are often present. Dysthymia is quite heterogeneous. Anxiety is not a necessary part of its clinical picture, yet dysthymia is often diagnosed in patients with anxiety and phobic disorders. The essential features of such primary dysthymia include habitual gloom, brooding, lack of joy in life, and preoccupation with inadequacy. Dysthymia then is best characterized as long-standing, fluctuating, low-grade depression, experienced as part of the habitual self and representing an accentuation of traits observed in the depressive temperament. The clinical picture of dysthymia is varied, with some patients proceeding to major depression and others manifesting the pathology largely at the personality level.

DIFFERENTIAL DIAGNOSIS:

The differential diagnosis for dysthymia is essentially identical to that for major depressive disorder. Many substances and medical illnesses can cause chronic depressive symptoms. Two disorders are particularly important to consider in the differential diagnosis of dysthymia—minor depressive disorder and recurrent brief depressive disorder.

COURSE AND PROGNOSIS:

About 50 percent of patients with dysthymia experience an insidious onset of symptoms before age 25 years. Despite the early onset, patients often suffer with the symptoms for a decade before seeking psychiatric help and may consider early-onset dysthymia simply part of life. Patients with an early onset of symptoms are at risk for either major depressive disorder or bipolar I disorder in the course of their disorder. Studies of patients with the diagnosis of dysthymia indicate that about 20 percent progressed to major depressive disorder, 15 percent to bipolar II disorder, and fewer than 5 percent to bipolar I

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disorder. The prognosis for patients with dysthymia varies. Antidepressive agents and specific types of psychotherapies (e.g., cognitive and behavior therapies) have positive effects on the course and prognosis of dysthymia. The available data about previously available treatments indicate that only 10 to 15 percent of patients are in remission 1 year after the initial diagnosis. About 25 percent of all patients with dysthymia never attain a complete recovery. Overall, however, the prognosis is good with treatment.

TREATMENT:

Historically, patients with dysthymia either received no treatment or were seen as candidates for long-term, insight-oriented psychotherapy. Contemporary data offer the most objective support for cognitive therapy, behavior therapy, and pharmacotherapy. The combination of pharmacotherapy and some form of psychotherapy may be the most effective treatment for the disorder.

4. CYCLOTHYMIC DISORDER

Cyclothymic disorder is symptomatically a mild form of bipolar II disorder, characterized by episodes of hypomania and mild depression. In DSM-5, cyclothymic disorder is defined as a “chronic, fluctuating mood disturbance” with many periods of hypomania and of depression. The disorder is differentiated from bipolar II disorder, which is characterized by the presence of major (not minor) depressive and hypomanic episodes.

EPIDEMIOLOGY:

Patients with cyclothymic disorder may constitute from 3 to 5 percent of all psychiatric outpatients, perhaps particularly those with significant complaints about marital and interpersonal difficulties. In the general population, the lifetime prevalence of cyclothymic disorder is estimated to be about 1 percent. This figure is probably lower than the actual prevalence because, as with patients with bipolar I disorder, the patients may not be aware that they have a psychiatric problem. Cyclothymic disorder, as with dysthymia, frequently coexists with borderline personality disorder. An estimated 10 percent of outpatients and 20 percent of inpatients with borderline personality disorder have a coexisting diagnosis of cyclothymic disorder. The female-to-male ratio in cyclothymic disorder is about 3 to 2, and 50 to 75 percent of all patients have an onset between ages 15 and 25 years. Families of persons with cyclothymic disorder often contain members with substance-related disorder.

ETIOLOGY:

Biological Factors-

About 30 percent of all patients with cyclothymic disorder have positive family histories for bipolar I disorder; this rate is similar to the rate for patients with bipolar I disorder. Moreover, the pedigrees of families with bipolar I disorder often contain generations of patients with bipolar I disorder linked by a generation with cyclothymic disorder. Conversely, the prevalence of cyclothymic disorder in the relatives of patients with bipolar I disorder is much higher than the prevalence of cyclothymic disorder either in the relatives of patients with other mental disorders or in persons who are mentally healthy.

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The observations that about one-third of patients with cyclothymic disorder subsequently have major mood disorders, that they are particularly sensitive to antidepressant-induced hypomania, and that about 60 percent respond to lithium add further support to the idea of cyclothymic disorder as a mild or attenuated form of bipolar II disorder.

Psychosocial Factors-

Most psychodynamic theories postulate that the development of cyclothymic disorder lies in traumas and fixations during the oral stage of infant development. Freud hypothesized that the cyclothymic state is the ego's attempt to overcome a harsh and punitive superego. Hypomania is explained psychodynamically as the lack of self-criticism and an absence of inhibitions occurring when a depressed person throws off the burden of an overly harsh superego. The major defense mechanism in hypomania is denial, by which the patient avoids external problems and internal feelings of depression. Patients with cyclothymic disorder are characterized by periods of depression alternating with periods of hypomania. Psychoanalytic exploration reveals that such patients defend themselves against underlying depressive themes with their euphoric or hypomanic periods. Hypomania is frequently triggered by a profound interpersonal loss. The false euphoria generated in such instances is a patient's way to deny dependence on love objects and simultaneously disavowing any aggression or destructiveness that may have contributed to the loss of the loved person.

DIAGNOSIS AND CLINICAL FEATURES:

The DSM-5 diagnostic criteria for cyclothymic disorder stipulate that a patient has never met the criteria for a major depressive episode and did not meet the criteria for a manic episode during the first 2 years of the disturbance. The criteria also require the more or less constant presence of symptoms for 2 years (or 1 year for children and adolescents).

Signs and Symptoms- The symptoms of cyclothymic disorder are identical to the symptoms of bipolar II disorder except that they are generally less severe. On occasion, however, the symptoms may be equally severe but of shorter duration than those seen in bipolar II disorder. About half of all patients with cyclothymic disorder have depression as their major symptom. Almost all patients with cyclothymic disorder have periods of mixed symptoms with marked irritability.

The lives of most patients with cyclothymic disorder are difficult. The cycles of the disorder tend to be much shorter than those in bipolar I disorder. In cyclothymic disorder, the changes in mood are irregular and abrupt and sometimes occur within hours. The unpredictable nature of the mood changes produces great stress. Patients often feel that their moods are out of control. In irritable, mixed periods, they may become involved in unprovoked disagreements with friends, family, and coworkers.

Substance Abuse- Alcohol abuse and other substance abuse are common in patients with cyclothymic disorder, who use substances either to self-medicate (with alcohol, benzodiazepines, and marijuana) or to achieve even further stimulation (with cocaine, amphetamines, and hallucinogens) when they are manic. About 5 to 10 percent of all patients with cyclothymic disorder have substance dependence.

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Persons with this disorder often have a history of multiple geographical moves, involvements in religious cults, and dilettantism.

DIFFERENTIAL DIAGNOSIS:

When a diagnosis of cyclothymic disorder is under consideration, all the possible medical and substance-related causes of depression and mania, such as seizures and particular substances (cocaine, amphetamine, and steroids), must be considered. Borderline, antisocial, histrionic, and narcissistic personality disorders should also be considered in the differential diagnosis.
Attention-deficit/hyperactivity disorder (ADHD) can be difficult to differentiate from cyclothymic disorder in children and adolescents. A trial of stimulants helps most patients with ADHD and exacerbates the symptoms of most patients with cyclothymic disorder. The diagnostic category of bipolar II disorder (discussed in Section 8.1) is characterized by the combination of major depressive and hypomanic episodes.

COURSE AND PROGNOSIS:

Some patients with cyclothymic disorder are characterized as having been sensitive, hyperactive, or moody as young children. The onset of frank symptoms of cyclothymic disorder often occurs insidiously in the teens or early 20s. The emergence of symptoms at that time hinders a person's performance in school and the ability to establish friendships with peers. The reactions of patients to such a disorder vary; patients with adaptive coping strategies or ego defenses have better outcomes than patients with poor coping strategies. About one-third of all patients with cyclothymic disorder develop a major mood disorder, most often bipolar II disorder.

TREATMENT:

Biological Therapy-

The mood stabilizers and antimanic drugs are the first line of treatment for patients with cyclothymic disorder. Although the experimental data are limited to studies with lithium, other antimanic agents—for example, carbamazepine and valproate (Depakene)—are reported to be effective. Dosages and plasma concentrations of these agents should be the same as those in bipolar I disorder. Antidepressant treatment of depressed patients with cyclothymic disorder should be done with caution because these patients have increased susceptibility to antidepressantinduced hypomanic or manic episodes. About 40 to 50 percent of all patients with cyclothymic disorder who are treated with antidepressants experience such episodes.

Psychosocial Therapy-

Psychotherapy for patients with cyclothymic disorder is best directed toward increasing patients' awareness of their condition and helping them develop coping mechanisms for their mood swings. Therapists usually need to help patients repair any damage, both work and family related, done during episodes of hypomania. Because of the long-term nature of cyclothymic disorder, patients often require lifelong treatment. Family and group therapies may be supportive, educational, and therapeutic for

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patients and for those involved in their lives. The psychiatrist conducting psychotherapy is able to evaluate the degree of cyclothymia and so provide an early-warning system to prevent full-blown manic attacks before they occur.

5. Major Depressive Disorder

Major Depressive Disorder, Single Episode-

Depression may occur as a single episode or may be recurrent. Differentiation between these patients and those who have two or more episodes of major depressive disorder is justified because of the uncertain course of the former patients' disorder. Several studies have reported data consistent with the notion that major depression covers a heterogeneous population of disorders. One type of study assessed the stability of a diagnosis of major depression in a patient over time. The study found that 25 to 50 percent of the patients were later reclassified as having a different psychiatric condition or a non-psychiatric medical condition with psychiatric symptoms. A second type of study evaluated first-degree relatives of affectively ill patients to determine the presence and types of psychiatric diagnoses for these relatives over time. Both types of studies found that depressed patients with more depressive symptoms are more likely to have stable diagnoses over time and are more likely to have affectively ill relatives than are depressed patients with fewer depressive symptoms. Also, patients with bipolar I disorder and those with bipolar II disorder (recurrent major depressive episodes with hypomania) are likely to have stable diagnoses over time.

Major Depressive Disorder, Recurrent-

Patients who are experiencing at least a second episode of depression are classified as having major depressive disorder, recurrent. The essential problem with diagnosing recurrent episodes of major depressive disorder is choosing the criteria to designate the resolution of each period. Two variables are the degree of resolution of the symptoms and the length of the resolution. DSM-5 requires that distinct episodes of depression be separated by at least 2 months during which a patient has no significant symptoms of depression.

COURSE:

ONSET-

About 50 percent of patients having their first episode of major depressive disorder exhibited significant depressive symptoms before the first identified episode. Therefore, early identification and treatment of early symptoms may prevent the development of a full depressive episode. Although symptoms may have been present, patients with major depressive disorder usually have not had a premorbid personality disorder. The first depressive episode occurs before age 40 years in about 50 percent of patients. A later onset is associated with the absence of a family history of mood disorders, antisocial personality disorder, and alcohol abuse.

DURATION-

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An untreated depressive episode lasts 6 to 13 months; most treated episodes last about 3 months. The withdrawal of antidepressants before 3 months has elapsed almost always results in the return of the symptoms. As the course of the disorder progresses, patients tend to have more frequent episodes that last longer. Over a 20- year period, the mean number of episodes is five or six.

DEVELOPMENT OF MANIC EPISODES-

About 5 to 10 percent of patients with an initial diagnosis of major depressive disorder have a manic episode 6 to 10 years after the first depressive episode. The mean age for this switch is 32 years, and it often occurs after two to four depressive episodes. Although the data are inconsistent and controversial, some clinicians report that the depression of patients who are later classified as having bipolar I disorder is often characterized by hypersomnia, psychomotor retardation, psychotic symptoms, a history of postpartum episodes, a family history of bipolar I disorder, and a history of antidepressant-induced hypomania.

PROGNOSIS:

Major depressive disorder is not a benign disorder. It tends to be chronic, and patients tend to relapse. Patients who have been hospitalized for a first episode of major depressive disorder have about a 50 percent chance of recovering in the first year. The percentage of patients recovering after repeated hospitalization decreases with passing time. Many unrecovered patients remain affected with dysthymic disorder. About 25 percent of patients experience a recurrence of major depressive disorder in the first 6 months after release from a hospital, about 30 to 50 percent in the following 2 years, and about 50 to 75 percent in 5 years. The incidence of relapse is lower than these figures in patients who continue prophylactic psychopharmacological treatment and in patients who have had only one or two depressive episodes. Generally, as a patient experiences more and more depressive episodes, the time between the episodes decreases, and the severity of each episode increases.

PROGNOSTIC INDICATORS-

Many studies have focused on identifying both good and bad prognostic indicators in the course of major depressive disorder. Mild episodes, the absence of psychotic symptoms, and a short hospital stay are good prognostic indicators. Psychosocial indicators of a good course include a history of solid friendships during adolescence, stable family functioning, and generally sound social functioning for the 5 years preceding the illness. Additional good prognostic signs are the absence of a comorbid psychiatric disorder and of a personality disorder, no more than one previous hospitalization for major depressive disorder, and an advanced age of onset. The possibility of a poor prognosis is increased by coexisting dysthymic disorder, abuse of alcohol and other substances, anxiety disorder symptoms, and a history of more than one previous depressive episode. Men are more likely than women to experience a chronically impaired course.

TREATMENT:

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The use of specific pharmacotherapy approximately doubles the chances that a depressed patient will recover in 1 month. All currently available antidepressants may take up to 3 to 4 weeks to exert significant therapeutic effects, although they may begin to show their effects earlier. Choice of antidepressants is determined by the side effect profile least objectionable to a given patient's physical status, temperament, and lifestyle. That numerous classes of antidepressants (Table 8.1- 10) are available, many with different mechanisms of action, represents indirect evidence for heterogeneity of putative biochemical lesions. Although the first antidepressant drugs, the monoamine oxidase inhibitors (MAOIs) and tricyclic antidepressants (TCAs), are still in use, newer compounds have made the treatment of depression more "clinician and patient friendly."

ANXIETY DISORDERS

PATHOLOGICAL ANXIETY

EPIDEMIOLOGY:

The anxiety disorders make up one of the most common groups of psychiatric disorders. The National Comorbidity Study reported that one of four persons met the diagnostic criteria for at least one anxiety disorder and that there is a 12-month prevalence rate of 17.7 percent. Women (30.5 percent lifetime prevalence) are more likely to have an anxiety disorder than are men (19.2 percent lifetime prevalence). The prevalence of anxiety disorders decreases with higher socioeconomic status.

1. Panic Disorder

An acute intense attack of anxiety accompanied by feelings of impending doom is known as panic disorder. The anxiety is characterized by discrete periods of intense fear that can vary from several attacks during one day to only a few attacks during a year. Patients with panic disorder present with a number of comorbid conditions, most commonly agoraphobia, which refers to a fear of or anxiety regarding places from which escape might be difficult.

EPIDEMIOLOGY:

The lifetime prevalence of panic disorder is in the 1 to 4 percent range, with 6-month prevalence approximately 0.5 to 1.0 percent and 3 to 5.6 percent for panic attacks. Women are two to three times more likely to be affected than men, although underdiagnosis of panic disorder in men may contribute to the skewed distribution. The differences among Hispanics, whites, and blacks are few. The only social factor identified as contributing to the development of panic disorder is a recent history of divorce or separation. Panic disorder most commonly develops in young adulthood—the mean age of presentation is about 25 years—but both panic disorder and agoraphobia can develop at any age. Panic disorder has been reported in children and adolescents, and it is probably underdiagnosed in these age groups.

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ETIOLOGY:

Biological Factors-

Considerable evidence indicates that abnormal regulation of brain noradrenergic systems is also involved in the pathophysiology of panic disorder. These and other studies have produced hypotheses implicating both peripheral and central nervous system (CNS) dysregulation in the pathophysiology of panic disorder. The autonomic nervous systems of some patients with panic disorder have been reported to exhibit increased sympathetic tone, to adapt slowly to repeated stimuli, and to respond excessively to moderate stimuli.

The major neurotransmitter systems that have been implicated are those for norepinephrine, serotonin, and GABA. Serotonergic dysfunction is quite evident in panic disorder, and various studies with mixed serotonin agonist–antagonist drugs have demonstrated increased rates of anxiety. Such responses may be caused by postsynaptic serotonin hypersensitivity in panic disorder. Preclinical evidence suggests that attenuation of local inhibitory GABAergic transmission in the basolateral amygdala, midbrain, and hypothalamus can elicit anxiety-like physiological responses. The biological data have led to a focus on the brainstem (particularly the noradrenergic neurons of the locus ceruleus and the serotonergic neurons of the median raphe nucleus), the limbic system (possibly responsible for the generation of anticipatory anxiety), and the prefrontal cortex (possibly responsible for the generation of phobic avoidance).

Genetic Factors-

Various studies have found that the first-degree relatives of patients with panic disorder have a four- to eightfold higher risk for panic disorder than first-degree relatives of other psychiatric patients. The twin studies conducted to date have generally reported that monozygotic twins are more likely to be concordant for panic disorder than are dizygotic twins. At this point, no data exist indicating an association between a specific chromosomal location or mode of transmission and this disorder.

Psychosocial Factors-

Psychoanalytic theories conceptualize panic attacks as arising from an unsuccessful defense against anxiety-provoking impulses. What was previously a mild signal anxiety becomes an overwhelming feeling of apprehension, complete with somatic symptoms . Although panic attacks are correlated neurophysiologically with the locus ceruleus, the onset of panic is generally related to environmental or psychological factors. Patients with panic disorder have a higher incidence of stressful life events (particularly loss) than control subjects in the months before the onset of panic disorder. Moreover, the patients typically experience greater distress about life events than control subjects do. The research indicates that the cause of panic attacks is likely to involve the unconscious meaning of stressful events and that the pathogenesis of the panic attacks may be related to neurophysiological factors triggered by the psychological reactions.

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DIAGNOSIS:

Panic Attacks-

A panic attack is a sudden period of intense fear or apprehension that may last from minutes to hours. Panic attacks can occur in mental disorders other than panic disorder, particularly in specific phobia, social phobia, and PTSD. Unexpected panic attacks occur at any time and are not associated with any identifiable situational stimulus, but panic attacks need not be unexpected. Attacks in patients with social and specific phobias are usually expected or cued to a recognized or specific stimulus. Some panic attacks do not fit easily into the distinction between unexpected and expected, and these attacks are referred to as situationally predisposed panic attacks. They may or may not occur when a patient is exposed to a specific trigger, or they may occur either immediately after exposure or after a considerable delay.

Panic Disorder-

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) diagnostic criteria for panic disorder are listed in Table 9.2-2. Some community surveys have indicated that panic attacks are common, and a major issue in developing diagnostic criteria for panic disorder was determining a threshold number or frequency of panic attacks required to meet the diagnosis. Setting the threshold too low results in the diagnosis of panic disorder in patients who do not have an impairment from an occasional panic attack; setting the threshold too high results in a situation in which patients who are impaired by their panic attacks do not meet the diagnostic criteria.

CLINICAL FEATURES:

The first panic attack is often completely spontaneous, although panic attacks occasionally follow excitement, physical exertion, sexual activity, or moderate emotional trauma. Clinicians should attempt to ascertain any habit or situation that commonly precedes a patient's panic attacks. Such activities may include the use of caffeine, alcohol, nicotine, or other substances; unusual patterns of sleeping or eating; and specific environmental settings, such as harsh lighting at work. The attack often begins with a 10-minute period of rapidly increasing symptoms. The major mental symptoms are extreme fear and a sense of impending death and doom. Patients usually cannot name the source of their fear; they may feel confused and have trouble concentrating. The physical signs often include tachycardia, palpitations, dyspnea, and sweating. Patients often try to leave whatever situation they are in to seek help. The attack generally lasts 20 to 30 minutes and rarely more than an hour. Patients may experience depression or depersonalization during an attack. The symptoms can disappear quickly or gradually. Between attacks, patients may have anticipatory anxiety about having another attack.

DIFFERENTIAL DIAGNOSIS:

Medical Disorders-

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Panic attacks are associated with a variety of endocrinological disorders, including both hypo- and hyperthyroid states, hyperparathyroidism, and pheochromocytomas. Episodic hypoglycemia associated with insulinomas can also produce panic-like states, as can primary neuropathological processes. These include seizure disorders, vestibular dysfunction, neoplasms, or the effects of both prescribed and illicit substances on the CNS. Finally, disorders of the cardiac and pulmonary systems, including arrhythmias, chronic obstructive pulmonary disease, and asthma, can produce autonomic symptoms and accompanying crescendo anxiety that can be difficult to distinguish from panic disorder.

Mental Disorders-

Panic attacks occur in many anxiety disorders, including social and specific phobia, Panic may also occur in PTSD and OCD. Differentiation from generalized anxiety disorder can also be difficult. Classically, panic attacks are characterized by their rapid onset (within minutes) and short duration (usually less than 10 to 15 minutes), in contrast to the anxiety associated with generalized anxiety disorder, which emerges and dissipates more slowly.

COURSE AND PROGNOSIS:

Panic disorder usually has its onset in late adolescence or early adulthood, although onset during childhood, early adolescence, and midlife does occur. About 30 to 40 percent of patients seem to be symptom free at long-term follow-up, about 50 percent have symptoms that are sufficiently mild not to affect their lives significantly, and about 10 to 20 percent continue to have significant symptoms. After the first one or two panic attacks, patients may be relatively unconcerned about their condition; with repeated attacks, however, the symptoms may become a major concern.

The frequency and severity of the attacks can fluctuate. Panic attacks can occur several times in a day or less than once a month. Excessive intake of caffeine or nicotine can exacerbate the symptoms. Depression can complicate the symptom picture in anywhere from 40 to 80 percent of all patients, as estimated by various studies. Although the patients do not tend to talk about suicidal ideation, they are at increased risk for committing suicide. Alcohol and other substance dependence occurs in about 20 to 40 percent of all patients, and OCD may also develop. Family interactions and performance in school and at work commonly suffer. Patients with good premorbid functioning and symptoms of brief duration tend to have good prognoses.

TREATMENT:

With treatment, most patients exhibit dramatic improvement in the symptoms of panic disorder and agoraphobia. The two most effective treatments are pharmacotherapy and cognitive-behavioral therapy. Family and group therapy may help affected patients and their families adjust to the patient's disorder and to the psychosocial difficulties that the disorder may have precipitated.

2. Specific Phobia

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The term phobia refers to an excessive fear of a specific object, circumstance, or situation. A specific phobia is a strong, persisting fear of an object or situation. The diagnosis of specific phobia requires the development of intense anxiety, even to the point of panic, when exposed to the feared object. Persons with specific phobias may anticipate harm, such as being bitten by a dog, or may panic at the thought of losing control; for instance, if they fear being in an elevator, they may also worry about fainting after the door closes.

EPIDEMIOLOGY:

Phobias are one of the most common mental disorders in the United States, where approximately 5 to 10 percent of the population is estimated to have these troubling and sometimes disabling disorders. The lifetime prevalence of specific phobia is about 10 percent. Specific phobia is the most common mental disorder among women and the second most common among men, second only to substance-related disorders. The 6- month prevalence of specific phobia is about 5 to 10 per 100 persons (Table 9.4-1). The rates of specific phobias in women (14 to 16 percent) were double those of men (5 to 7 percent), although the ratio is closer to 1 to 1 for the fear of blood, injection, or injury type. (Types of phobias are discussed below in this section.) The peak age of onset for the natural environment type and the blood-injection-injury type is in the range of 5 to 9 years, although onset also occurs at older ages. In contrast, the peak age of onset for the situational type (except fear of heights) is higher, in the mid-20s, which is closer to the age of onset for agoraphobia. The feared objects and situations in specific phobias (listed in descending frequency of appearance) are animals, storms, heights, illness, injury, and death.

ETIOLOGY:

-Behavioral Factors

-Psychoanalytic Factors

Phobias illustrate the interaction between a genetic constitutional diathesis and environmental stressors. Longitudinal studies suggest that certain children are constitutionally predisposed to phobias because they are born with a specific temperament known as behavioral inhibition to the unfamiliar, but a chronic environmental stress must act on a child's temperamental disposition to create a fullblown phobia. Stressors, such as the death of a parent, separation from a parent, criticism or humiliation by an older sibling, and violence in the household, may activate the latent diathesis within the child, who then becomes symptomatic.

DIAGNOSIS:

The DSM-5 includes distinctive types of specific phobia: animal type, natural environment type (e.g., storms), blood-injection-injury type (e.g., needles), situational type (e.g., cars, elevators, planes), and other type (for specific phobias that do not fit into the previous four types). The key feature of each type of phobia is that fear symptoms occur only in the presence of a specific object (Table 9.4-3). The bloodinjection-injury type is differentiated from the others in that bradycardia and

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hypotension often follow the initial tachycardia that is common to all phobias. The blood-injection-injury type of specific phobia is particularly likely to affect many members and generations of a family. One type of phobia of recently reported phobia is space phobia, in which persons fear falling when there is no nearby support, such as a wall or a chair. Some data indicate that affected persons may have abnormal right hemisphere function, possibly resulting in visual-spatial impairment. Balance disorders should be ruled out in such patients.

CLINICAL FEATURES:

Phobias are characterized by the arousal of severe anxiety when patients are exposed to specific situations or objects or when patients even anticipate exposure to the situations or objects. Exposure to the phobic stimulus or anticipation of it almost invariably results in a panic attack in a person who is susceptible to them. Persons with phobias, by definition, try to avoid the phobic stimulus; some go to great trouble to avoid anxiety-provoking situations. For example, a patient with a phobia may take a bus across the United States, rather than fly, to avoid contact with the object of the patient's phobia, an airplane. Perhaps as another way to avoid the stress of the phobic stimulus, many patients have substance-related disorders, particularly alcohol use disorders. Moreover, an estimated one-third of patients with social phobia have major depressive disorder. The major finding on the mental status examination is the presence of an irrational and ego-dystonic fear of a specific situation, activity, or object; patients are able to describe how they avoid contact with the phobia. Depression is commonly found on the mental status examination and may be present in as many as one-third of all patients with phobia.

DIFFERENTIAL DIAGNOSIS:

Nonpsychiatric medical conditions that can result in the development of a phobia include the use of substances (particularly hallucinogens and sympathomimetics), CNS tumors, and cerebrovascular diseases. Phobic symptoms in these instances are unlikely in the absence of additional suggestive findings on physical, neurological, and mental status examinations. Schizophrenia is also in the differential diagnosis of specific phobia because patients with schizophrenia can have phobic symptoms as part of their psychoses. Unlike patients with schizophrenia, however, patients with phobia have insight into the irrationality of their fears and lack the bizarre quality and other psychotic symptoms that accompany schizophrenia. In the differential diagnosis of specific phobia, clinicians must consider panic disorder, agoraphobia, and avoidant personality disorder. Differentiation among panic disorder, agoraphobia, social phobia, and specific phobia can be difficult in individual cases. In general, however, patients with specific phobia tend to experience anxiety immediately when presented with the phobic stimulus. Furthermore, the anxiety or panic is limited to the identified situation; patients are not abnormally anxious when they are neither confronted with the phobic stimulus nor caused to anticipate the stimulus. Other diagnoses to consider in the differential diagnosis of specific phobia are hypochondriasis, OCD, and paranoid personality disorder. Whereas hypochondriasis is the fear of having a disease, specific phobia of the illness type is the fear of contracting the disease. Some patients with OCD manifest behavior indistinguishable from that of a patient with specific phobia. For example, whereas patients with OCD may avoid

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knives because they have compulsive thoughts about killing their children, patients with specific phobia about knives may avoid them for fear of cutting themselves. Patients with paranoid personality disorder have generalized fear that distinguishes them from those with specific phobia.

COURSE AND PROGNOSIS:

Specific phobia exhibits a bimodal age of onset, with a childhood peak for animal phobia, natural environment phobia, and blood-injection-injury phobia and an early adulthood peak for other phobias, such as situational phobia. Limited prospective epidemiological data are available that chart the natural course of specific phobia. Because patients with isolated specific phobia rarely present for treatment, there is also little research on the course of the disorder in the clinic. The limited information that is available suggests that most specific phobias that begin in childhood and persist into adulthood will continue to persist for many years. The severity of the condition is believed to remain relatively constant, which contrasts with the waxing and waning course seen in other anxiety disorders.

TREATMENT:

- Behavior Therapy

- Insight-Oriented Psychotherapy

- Virtual Therapy

Specific Phobia-

A common treatment for specific phobia is exposure therapy. In this method, therapists desensitize patients by using a series of gradual, self-paced exposures to the phobic stimuli, and they teach patients various techniques to deal with anxiety, including relaxation, breathing control, and cognitive approaches. The cognitive-behavioral approaches include reinforcing the realization that the phobic situation is, in fact, safe. The key aspects of successful behavior therapy are the patient's commitment to treatment, clearly identified problems and objectives, and alternative strategies for coping with the patient's feelings. In the special situation of blood-injection-injury phobia, some therapists recommend that patients tense their bodies and remain seated during the exposure to help avoid the possibility of fainting from a vasovagal reaction to the phobic stimulation. β-adrenergic receptor antagonists may be useful in the treatment of specific phobia, especially when the phobia is associated with panic attacks. Pharmacotherapy (e.g., benzodiazepines), psychotherapy, or combined therapy directed to the attacks may also be of benefit.

3. Generalized Anxiety Disorder

Generalized anxiety disorder is defined as excessive anxiety and worry about several events or activities for most days during at least a 6-month period. The worry is difficult to control and is associated with somatic symptoms, such as muscle tension, irritability, difficulty sleeping, and restlessness.

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EPIDEMIOLOGY:

Generalized anxiety disorder is a common condition; reasonable estimates for its 1-year prevalence range from 3 to 8 percent. The ratio of women to men with the disorder is about 2 to 1, but the ratio of women to men who are receiving inpatient treatment for the disorder is about 1 to 1. A lifetime prevalence is close to 5 percent with the Epidemiological Catchment Area (ECA) study suggesting a lifetime prevalence as high as 8 percent. In anxiety disorder clinics, about 25 percent of patients have generalized anxiety disorder. The disorder usually has its onset in late adolescence or early adulthood, although cases are commonly seen in older adults. Also, some evidence suggests that the prevalence of generalized anxiety disorder is particularly high in primary care settings.

ETIOLOGY:

Biological Factors-

benzodiazepines and the azaspirones (e.g., buspirone [BuSpar]) have focused biological research efforts on the γ-aminobutyric acid and serotonin neurotransmitter systems. Whereas benzodiazepines (which are benzodiazepine receptor agonists) are known to reduce anxiety, flumazenil (Romazicon) (a benzodiazepine receptor antagonist) and the β-carbolines (benzodiazepine receptor reverse agonists) are known to induce anxiety.

Brain areas hypothesized to be involved in generalized anxiety disorder are the basal ganglia, the limbic system, and the frontal cortex. regulation of the serotonergic system in generalized anxiety disorder is abnormal. Other neurotransmitter systems that have been the subject of research in generalized anxiety disorder include the norepinephrine, glutamate, and cholecystokinin systems. Some evidence indicates that patients with generalized anxiety disorder may have subsensitivity of their α2 -adrenergic receptors. PET study reported a lower metabolic rate in basal ganglia and white matter in patients with generalized anxiety disorder than in normal control subjects.

Psychosocial Factors-

The two major schools of thought about psychosocial factors leading to the development of generalized anxiety disorder are the cognitive-behavioral school and the psychoanalytic school. According to the cognitive-behavioral school, patients with generalized anxiety disorder respond to incorrectly and inaccurately perceived dangers. The inaccuracy is generated by selective attention to negative details in the environment, by distortions in information processing, and by an overly negative view of the person's own ability to cope. The psychoanalytic school hypothesizes that anxiety is a symptom of unresolved, unconscious conflicts.

DIAGNOSIS:

Generalized anxiety disorder is characterized by a pattern of frequent, persistent worry and anxiety that is out of proportion to the impact of the event or circumstance that is the focus of the worry. The distinction between generalized anxiety disorder and normal anxiety is emphasized by the use

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of the word “excessive” in the criteria and by the specification that the symptoms cause significant impairment or distress.

CLINICAL FEATURES:

The essential characteristics of generalized anxiety disorder are sustained and excessive anxiety and worry accompanied by either motor tension or restlessness. The anxiety is excessive and interferes with other aspects of a person’s life. This pattern must occur more days than not for at least 3 months. The motor tension is most commonly manifested as shakiness, restlessness, and headaches. Patients with generalized anxiety disorder usually seek out a general practitioner or internist for help with a somatic symptom. Alternatively, the patients go to a specialist for a specific symptom (e.g., chronic diarrhea). A specific nonpsychiatric medical disorder is rarely found, and patients vary in their doctor-seeking behavior. Some patients accept a diagnosis of generalized anxiety disorder and the appropriate treatment; others seek additional medical consultations for their problems.

DIFFERENTIAL DIAGNOSIS:

As with other anxiety disorders, generalized anxiety disorder must be differentiated from both medical and psychiatric disorders. Neurological, endocrinological, metabolic, and medication-related disorders similar to those considered in the differential diagnosis of panic disorder must be considered in the differential diagnosis of generalized anxiety disorder. Common co-occurring anxiety disorders also must be considered, including panic disorder, phobias, OCD, and PTSD. To meet criteria for generalized anxiety disorder, patients must both exhibit the full syndrome, and their symptoms also cannot be explained by the presence of a comorbid anxiety disorder. To diagnose generalized anxiety disorder in the context of other anxiety disorders, it is most important to document anxiety or worry related to circumstances or topics that are either unrelated, or only minimally related, to other disorders. Proper diagnosis involves both definitively establishing the presence of generalized anxiety disorder and properly diagnosing other anxiety disorders. Patients with generalized anxiety disorder frequently develop major depressive disorder. As a result, this condition must also be recognized and distinguished. The key to making a correct diagnosis is documenting anxiety or worry that is unrelated to the depressive disorder.

COURSE AND PROGNOSIS:

The age of onset is difficult to specify; most patients with the disorder report that they have been anxious for as long as they can remember. Patients usually come to a clinician’s attention in their 20s, although the first contact with a clinician can occur at virtually any age. Only one-third of patients who have generalized anxiety disorder seek psychiatric treatment. Many go to general practitioners, internists, cardiologists, pulmonary specialists, or gastroenterologists, seeking treatment for the somatic component of the disorder. Because of the high incidence of comorbid mental disorders in patients with generalized anxiety disorder, the clinical course and prognosis of the disorder are difficult to predict. Nonetheless, some data indicate that life events are associated with the onset of generalized anxiety disorder: The occurrence of several negative life events greatly

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increases the likelihood that the disorder will develop. By definition, generalized anxiety disorder is a chronic condition that may well be lifelong.

TREATMENT:

The most effective treatment of generalized anxiety disorder is probably one that combines psychotherapeutic, pharmacotherapeutic, and supportive approaches. The treatment may take a significant amount of time for the involved clinician, whether the clinician is a psychiatrist, a family practitioner, or another specialist.

Obsessive-Compulsive and Related Disorders

1. Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) is represented by a diverse group of symptoms that include intrusive thoughts, rituals, preoccupations, and compulsions. These recurrent obsessions or compulsions cause severe distress to the person. The obsessions or compulsions are time-consuming and interfere significantly with the person's normal routine, occupational functioning, usual social activities, or relationships. A patient with OCD may have an obsession, a compulsion, or both. An obsession is a recurrent and intrusive thought, feeling, idea, or sensation. In contrast to an obsession, which is a mental event, a compulsion is a behavior. Specifically, a compulsion is a conscious, standardized, recurrent behavior, such as counting, checking, or avoiding.

EPIDEMIOLOGY:

The rates of OCD are fairly consistent, with a lifetime prevalence in the general population estimated at 2 to 3 percent. These figures make OCD the fourth most common psychiatric diagnosis after phobias, substance-related disorders, and major depressive disorder. Epidemiological studies in Europe, Asia, and Africa have confirmed these rates across cultural boundaries. Among adults, men and women are equally likely to be affected, but among adolescents, boys are more commonly affected than girls. The mean age of onset is about 20 years, although men have a slightly earlier age of onset (mean about 19 years) than women (mean about 22 years). Overall, the symptoms of about two thirds of affected persons have an onset before age 25, and the symptoms of fewer than 15 percent have an onset after age 35. The onset of the disorder can occur in adolescence or childhood, in some cases as early as 2 years of age.

ETIOLOGY:

Biological Factors-

Neurotransmitters –

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SEROTONERGIC SYSTEM- The many clinical drug trials that have been conducted support the hypothesis that dysregulation of serotonin is involved in the symptom formation of obsessions and compulsions in the disorder.

NORADRENERGIC SYSTEM- Currently, less evidence exists for dysfunction in the noradrenergic system in OCD.

Genetics-

Available genetic data on OCD support the hypothesis that the disorder has a significant genetic component. Relatives of probands with OCD consistently have a threefold to fivefold higher probability of having OCD or obsessive-compulsive features than families of control probands.

Psychosocial Factors-

Personality Factors- OCD differs from obsessive-compulsive personality disorder, which is associated with an obsessive concern for details, perfectionism, and other similar personality traits. Most persons with OCD do not have premorbid compulsive symptoms, and such personality traits are neither necessary nor sufficient for the development of OCD. Only about 15 to 35 percent of patients with OCD have had premorbid obsessional traits.

Psychodynamic Factors- Psychodynamic insight may be of great help in understanding problems with treatment compliance, interpersonal difficulties, and personality problems.

Finally, one other contribution of psychodynamic thinking is recognition of the precipitants that initiate or exacerbate symptoms. Often, interpersonal difficulties increase the patient's anxiety and, thus, increase the patient's symptomatology as well. Research suggests that OCD may be precipitated by a number of environmental stressors, especially those involving pregnancy, childbirth, or parental care of children. An understanding of the stressors may assist the clinician in an overall treatment plan that reduces the stressful events themselves or their meaning to the patient.

DIAGNOSIS AND CLINICAL FEATURES:

As part of the diagnostic criteria for OCD, the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) allows clinicians to indicate whether the patient's OCD is characterized by good or fair insight, poor insight, or absent insight (Table 10.1- 1). Patients with good or fair insight recognize that their OCD beliefs are definitely or probably not true or may or may not be true. Patients with poor insight believe their OCD beliefs are probably true, and patients with absent insight are convinced that their beliefs are true.

Patients with OCD often take their complaints to physicians rather than psychiatrists. Most patients with OCD have both obsessions and compulsions—up to 75 percent in some surveys. Some

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researchers and clinicians believe that the number may be much closer to 100 percent if patients are carefully assessed for the presence of mental compulsions in addition to behavioral compulsions. For example, an obsession about hurting a child may be followed by a mental compulsion to repeat a specific prayer a specific number of times. Other researchers and clinicians, however, believe that some patients do have only obsessive thoughts without compulsions. Such patients are likely to have repetitious thoughts of a sexual or aggressive act that is reprehensible to them. For clarity, it is best to conceptualize obsessions as thoughts and compulsions as behavior.

A feeling of anxious dread accompanies the central manifestation, and the key characteristic of a compulsion is that it reduces the anxiety associated with the obsession. The obsession or the compulsion is ego-alien; that is, it is experienced as foreign to the person's experience of himself or herself as a psychological being. No matter how vivid and compelling the obsession or compulsion, the person usually recognizes it as absurd and irrational. The person suffering from obsessions and compulsions usually feels a strong desire to resist them. Nevertheless, about half of all patients offer little resistance to compulsions, although about 80 percent of all patients believe that the compulsion is irrational. Sometimes, patients overvalue obsessions and compulsions—for example, they may insist that compulsive cleanliness is morally correct, even though they have lost their jobs because of time spent cleaning.

DIFFERENTIAL DIAGNOSIS:

Medical Conditions - Sydenham's chorea and Huntington's disease.

OCD is closely related to Tourette's disorder, as the two conditions frequently co-occur, both in individuals over time and within families. About 90 percent of persons with Tourette's disorder have compulsive symptoms, and as many as two thirds meet the diagnostic criteria for OCD.

OCD exhibits a superficial resemblance to obsessive-compulsive personality disorder, which is associated with an obsessive concern for details, perfectionism, and other similar personality traits. The conditions are easily distinguished in that only OCD is associated with a true syndrome of obsessions and compulsions.

The keys to distinguishing OCD from psychosis are (1) patients with OCD can almost always acknowledge the unreasonable nature of their symptoms, and (2) psychotic illnesses are typically associated with a host of other features that are not characteristic of OCD. Similarly, OCD can be difficult to differentiate from depression because the two disorders often occur comorbidly, and major depression is often associated with obsessive thoughts that, at times, border on true obsessions such as those that characterize OCD. The two conditions are best distinguished by their courses. Obsessive symptoms associated with depression are only found in the presence of a depressive episode, whereas true OCD persists despite remission of depression.

COURSE AND PROGNOSIS:

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More than half of patients with OCD have a sudden onset of symptoms. The onset of symptoms for about 50 to 70 percent of patients occurs after a stressful event, such as a pregnancy, a sexual problem, or the death of a relative. Because many persons manage to keep their symptoms secret, they often delay 5 to 10 years before coming to psychiatric attention, although the delay is probably shortening with increased awareness of the disorder. The course is usually long but variable; some patients experience a fluctuating course, and others experience a constant one. About 20 to 30 percent of patients have significant improvement in their symptoms, and 40 to 50 percent have moderate improvement. The remaining 20 to 40 percent of patients either remain ill or their symptoms worsen. About one-third of patients with OCD have major depressive disorder, and suicide is a risk for all patients with OCD. A poor prognosis is indicated by yielding to (rather than resisting) compulsions, childhood onset, bizarre compulsions, the need for hospitalization, a coexisting major depressive disorder, delusional beliefs, the presence of overvalued ideas (i.e., some acceptance of obsessions and compulsions), and the presence of a personality disorder (especially schizotypal personality disorder). A good prognosis is indicated by good social and occupational adjustment, the presence of a precipitating event, and an episodic nature of the symptoms. The obsessional content does not seem to be related to the prognosis.

TREATMENT:

With mounting evidence that OCD is largely determined by biological factors, classic psychoanalytic theory has fallen out of favor. Moreover, because OCD symptoms appear to be largely refractory to psychodynamic psychotherapy and psychoanalysis, pharmacological and behavioral treatments have become common. But psychodynamic factors may be of considerable benefit in understanding what precipitates exacerbations of the disorder and in treating various forms of resistance to treatment, such as noncompliance with medication. Many patients with OCD tenaciously resist treatment efforts. They may refuse to take medication and may resist carrying out therapeutic homework assignments and other activities prescribed by behavior therapists. The obsessive-compulsive symptoms themselves, no matter how biologically based, may have important psychological meanings that make patients reluctant to give them up. Psychodynamic exploration of a patient's resistance to treatment may improve compliance. Well-controlled studies have found that pharmacotherapy, behavior therapy, or a combination of both is effective in significantly reducing the symptoms of patients with OCD. The decision about which therapy to use is based on the clinician's judgment and experience and the patient's acceptance of the various modalities.

2. Body Dysmorphic Disorder

Body dysmorphic disorder is characterized by a preoccupation with an imagined defect in appearance that causes clinically significant distress or impairment in important areas of functioning. If a slight physical anomaly is actually present, the person's concern with the anomaly is excessive and bothersome.

EPIDEMIOLOGY:

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Body dysmorphic disorder is a poorly studied condition, partly because patients are more likely to go to dermatologists, internists, or plastic surgeons than to psychiatrists for this condition. One study of a group of college students found that more than 50 percent had at least some preoccupation with a particular aspect of their appearance, and in about 25 percent of the students, the concern had at least some significant effect on their feelings and functioning. DSM-5 reports a point prevalence in the United States of 2.4 percent. Available data indicate that the most common age of onset is between 15 and 30 years and that women are affected somewhat more often than men. Affected patients are also likely to be unmarried. Body dysmorphic disorder commonly coexists with other mental disorders. One study found that more than 90 percent of patients with body dysmorphic disorder had experienced a major depressive episode in their lifetimes; about 70 percent had experienced an anxiety disorder; and about 30 percent had experienced a psychotic disorder.

ETIOLOGY:

The cause of body dysmorphic disorder is unknown. The high comorbidity with depressive disorders, a higher-than-expected family history of mood disorders and OCD, and the reported responsiveness of the condition to serotonin-specific drugs indicate that, in at least some patients, the pathophysiology of the disorder may involve serotonin and may be related to other mental disorders. Stereotyped concepts of beauty emphasized in certain families and within the culture at large may significantly affect patients with body dysmorphic disorder. In psychodynamic models, body dysmorphic disorder is seen as reflecting the displacement of a sexual or emotional conflict onto a nonrelated body part. Such an association occurs through the defense mechanisms of repression, dissociation, distortion, symbolization, and projection.

DIAGNOSIS:

The DSM-5 diagnostic criteria for body dysmorphic disorder stipulate preoccupation with a perceived defect in appearance or overemphasis of a slight defect. It also stipulates that at some point during the course of the disorder, the patient performs compulsive behaviors (i.e., mirror checking, excessive grooming) or mental acts (e.g., comparing their appearance to that of others). The preoccupation causes patients significant emotional distress or markedly impairs their ability to function in important areas.

CLINICAL FEATURES:

The most common concerns (Table 10.2-1) involve facial flaws, particularly those involving specific parts (e.g., the nose). Sometimes the concern is vague and difficult to understand, such as extreme concern over a “scrunchy” chin. One study found that, on average, patients had concerns about four body regions during the course of the disorder. Other body parts of concern are hair, breasts, and genitalia. A proposed variant of dysmorphic disorder among men is the desire to “bulk up” and develop large muscle mass, which can interfere with ordinary living, holding a job, or staying healthy. The specific body part may change during the time a patient is affected with the disorder. Common associated symptoms include ideas or frank delusions of reference (usually about persons’

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noticing the alleged body flaw), either excessive mirror checking or avoidance of reflective surfaces, and attempts to hide the presumed deformity (with makeup or clothing). The effects on a person's life can be significant; almost all affected patients avoid social and occupational exposure. As many as one-third of patients may be housebound because of worry about being ridiculed for the alleged deformities; and approximately one-fifth of patients attempt suicide. As discussed, comorbid diagnoses of depressive disorders and anxiety disorders are common, and patients may also have traits of OCD, schizoid, and narcissistic personality disorders.

DIFFERENTIAL DIAGNOSIS:

The diagnosis of body dysmorphic disorder should not be made if the excessive bodily preoccupation is better accounted for by another psychiatric disorder. Excessive bodily preoccupation is generally restricted to concerns about being fat in anorexia nervosa; to discomfort with, or a sense of wrongness about, his or her primary and secondary sex characteristics occurring in gender identity disorder; and to mood-congruent cognitions involving appearance that occur exclusively during a major depressive episode. Individuals with avoidant personality disorder or social phobia may worry about being embarrassed by imagined or real defects in appearance, but this concern is usually not prominent, persistent, distressing, or impairing. Taijin kyofusho, a diagnosis in Japan, is similar to social phobia but has some features that are more consistent with body dysmorphic disorder, such as the belief that the person has an offensive odor or body parts that are offensive to others. Although individuals with body dysmorphic disorder have obsessional preoccupations about their appearance and may have associated compulsive behaviors (e.g., mirror checking), a separate or additional diagnosis of OCD is made only when the obsessions or compulsions are not restricted to concerns about appearance and are ego-dystonic. An additional diagnosis of delusional disorder, somatic type, can be made in people with body dysmorphic disorder only if their preoccupation with the imagined defect in appearance is held with a delusional intensity. Unlike normal concerns about appearance, the preoccupation with appearance and specific imagined defects in body dysmorphic disorder and the changed behavior because of the preoccupation are excessively time-consuming and are associated with significant distress or impairment.

COURSE AND PROGNOSIS:

Body dysmorphic disorder usually begins during adolescence, although it may begin later after a protracted dissatisfaction with the body. Age of onset is not well understood because variably a long delay occurs between symptom onset and treatment seeking. The onset can be gradual or abrupt. The disorder usually has a long and undulating course with few symptom-free intervals. The part of the body on which concern is focused may remain the same or may change over time.

TREATMENT:

Treatment of patients with body dysmorphic disorder with surgical, dermatological, dental, and other medical procedures to address the alleged defects is almost invariably unsuccessful. Although tricyclic drugs, monoamine oxidase inhibitors (MAOIs), and pimozide (Orap) have reportedly been

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useful in individual cases, other data indicate that serotonin-specific drugs—for example, clomipramine (Anafranil) and fluoxetine (Prozac)—reduce symptoms in at least 50 percent of patients. In any patient with a coexisting mental disorder, such as a depressive disorder or an anxiety disorder, the coexisting disorder should be treated with the appropriate pharmacotherapy and psychotherapy. How long treatment should be continued after the symptoms of body dysmorphic disorder have remitted is unknown. Augmentation of the selective serotonin reuptake inhibitor (SSRI) with clomipramine (Anafranil), buspirone (BuSpar), lithium (Eskalith), methylphenidate (Ritalin), or antipsychotics may improve the response rate.

3. Hoarding Disorder

Compulsive hoarding is a common and often disabling phenomenon associated with impairment in such functions as eating, sleeping, and grooming. Hoarding may result in health problems and poor sanitation, particularly when hoarding of animals is involved, and may lead to death from fire or falling. The disorder is characterized by acquiring and not discarding things that are deemed to be of little or no value, resulting in excessive clutter of living spaces. Hoarding was originally considered a subtype of obsessive-compulsive disorder (OCD), but is now considered to be a separate diagnostic entity. It is commonly driven by an obsessive fear of losing important items that the person believes may be of use at some point in the future, by distorted beliefs about the importance of possessions, and by extreme emotional attachment to possessions.

EPIDEMIOLOGY:

Hoarding is believed to occur in approximately 2 to 5 percent of the population, although some studies have found lifetime prevalence as high as 14 percent. It occurs equally among men and women, is more common in single persons, and is associated with social anxiety, withdrawal, and dependent personality traits. Hoarding usually begins in early adolescence and persists throughout the lifespan.

ETIOLOGY:

Little is known about the etiology of hoarding disorder. Research has shown a familial aspect to hoarding disorder, with about 80 percent of hoarders reporting at least one first-degree relative with hoarding behavior. Biological research has shown a lower metabolism in the posterior cingulate cortex and the occipital cortex of hoarders, which may also account for various cognitive impairments within hoarders such as attention and decision-making deficits. One study of the molecular genetics for hoarding found a link between hoarding behavior and markers on chromosomes 4q, 5q, and17q. Another study found that the catecholamine-O-methyltransferase (COMT) gene on chromosome 22q11.21 might contribute to the genetic susceptibility to hoarding.

DIAGNOSIS:

Hoarding disorder is characterized by (1) the acquiring of and failure to discard a large amount of possessions that are deemed useless or of little value; (2) greatly cluttered living areas precluding

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normal activities; and (3) significant distress and impairment in functioning due to hoarding. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) includes diagnostic specifiers that relate to insight, which may be rated poor, fair, or good. Some patients are completely unaware of the full extent of the problem and totally resistant to treatment. At times, delusional beliefs about hoarded items are present.

CLINICAL FEATURES:

Hoarding is driven by the fear of losing items that the patient believes will be needed later and a distorted belief about or an emotional attachment to possessions. Most hoarders do not perceive their behavior to be a problem. In fact, many perceive their behavior to be reasonable and part of their identity. Most hoarding patients accumulate possessions passively rather than intentionally, thus clutter accumulates gradually over time. Common hoarded items include newspapers, mail, magazines, old clothes, bags, books, lists, and notes. Hoarding poses risks to not only the patient, but also to those around them. Clutter accumulated from hoarding has been attributed to deaths from fire or patients being crushed by their possessions. It can also attract pest infestations that can pose a health risk both to the patient and residents around them. Many sufferers have been evicted from their home or threatened with eviction as a result of their hoarding. In severe cases, hoarding can interfere with work, social interaction, and basic activities such as eating or sleeping. The pathological nature of hoarding comes from the inability to organize possessions and keep them organized. Many hoard to avoid making decisions about discarding items. Patients with hoarding disorder also overemphasize the importance of recalling information and possessions. For example, a hoarder will keep old newspapers and magazines because they believe that if discarded the information will be forgotten and will never be retrieved again. In addition, patients believe that forgetting information will lead to serious consequences and prefer to keep possessions in sight so as not to forget them.

DIFFERENTIAL DIAGNOSIS:

The diagnosis of hoarding disorder should not be made if the excessive acquisition and inability to discard possessions is better accounted for by another medical or psychiatric condition. Until recently, hoarding was considered to be a symptom of OCD and obsessive-compulsive personality disorder. However, there are some major differences. Hoarding disorder patients do not display some of the classic symptoms of OCD such as recurring intrusive thoughts or compulsive rituals. Unlike symptoms of OCD, symptoms of hoarding worsen with time, rituals are not fixed, and obsessions about dirt or contamination are absent. OCD patients have better insight into their condition. Symptoms are usually ego-dystonic, whereas in hoarding disorder they are ego-syntonic. Hoarding behavior is seldom repetitive and is not viewed as intrusive or distressing to the hoarder. Distress mainly comes at the prospect of discarding items, and it manifests more as guilt and anger than anxiety. Hoarding disorder also tends to be less responsive to classic treatments for OCD such as exposure therapy, cognitive-behavioral therapy (CBT), and selective serotonin reuptake inhibitors (SSRIs). Some case reports show the onset of this behavior in patients after suffering brain lesions. Hoarding associated with brain lesions is more purposeless than hoarding that is motivated by

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emotional attachment or high intrinsic value of possessions. It is a common symptom in moderate to severe dementia. In cases of dementia, hoarding is often associated with a higher prevalence of hiding, rummaging, repetitive behavior, pilfering, and hyperphagia. Onset of the behavior usually coincides with onset of the dementia, starting in an organized manner, and becomes more disorganized as the disease progresses. The onset of dementia in a patient who has hoarded throughout his or her lifetime can aggravate the hoarding behavior. Hoarding behavior can be associated with schizophrenia. It is mostly associated with severe cases and is seen as a repetitive behavior associated with delusions, self-neglect, and squalor. Bipolar disorder is ruled out by the absence of severe mood swings.

COURSE AND PROGNOSIS:

The disorder is a chronic condition with a treatment-resistant course. Treatment seeking does not usually occur until patients are in their 40s or 50s, even if the hoarding began during adolescence. Symptoms may fluctuate throughout the course of the disorder, but full remission is rare. Patients have very little insight into their behavior and usually seek treatment under pressure from others. Some patients begin hoarding in response to a stressful event, while others report a slow and steady progression throughout life. Those who report onset due to a stressful event have a later age of onset than those who do not. Those with an earlier age of onset run a longer and more chronic course.

TREATMENT:

Hoarding disorder is difficult to treat. Although it shows similarities to OCD, effective treatments for OCD have shown little benefit for patients with hoarding disorder. In one study, only 18 percent of patients responded to medication and CBT. The challenges posed by hoarding patients to typical CBT treatment include poor insight to the behavior and low motivation and resistance to treatment. The most effective treatment for the disorder is a cognitive behavioral model that includes training in decision making and categorizing; exposure and habituation to discarding; and cognitive restructuring. This includes both office and in-home sessions. The role of the therapist in this model is to assist in the development of decision-making skills, to provide feedback about normal saving behavior, and to identify and challenge the patient's erroneous beliefs about possessions. The goal in treatment is to get rid of a significant amount of possessions, thereby making the living space livable, and to provide the patient with the skills to maintain a positive balance between the amount of possessions and livable space. Studies have shown a 25 to 34 percent reduction in hoarding behaviors using this method. Restructuring of this method for group and webbased interventions are currently under study and show promise. Pharmacological treatment studies using SSRIs have shown mixed results. Some studies have shown a negative response to SSRI treatment in hoarding patients compared with nonhoarders, while others have found no significant difference between the two groups.

4. **Hair-Pulling Disorder (Trichotillomania)**

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Hair-pulling disorder is a chronic disorder characterized by repetitive hair pulling, leading to variable hair loss that may be visible to others. It is also known as trichotillomania, a term coined by a French dermatologist Francois Hallopeau in 1889. The disorder was once deemed rare and little about it was described beyond phenomenology. It is now regarded as more common. The disorder is similar to obsessive-compulsive disorder and impulse control in that there is increased tension prior to the hair pulling and a relief of tension or gratification after the hair pulling.

EPIDEMIOLOGY:

The prevalence of hair-pulling disorder may be underestimated because of accompanying shame and secretiveness. The diagnosis encompasses at least two categories of hair pulling that differ in incidence, severity, age of presentation, and gender ratio. Other subsets may exist. The most serious, chronic form of the disorder usually begins in early to mid adolescence, with a lifetime prevalence ranging from 0.6 percent to as high as 3.4 percent in general populations and with female to male ratio as high as 10 to 1. The number of men may actually be higher, because men are even more likely than women to conceal hair pulling. A patient with chronic hair-pulling disorder is likely to be the only or oldest child in the family. A childhood type of hair-pulling disorder occurs approximately equally in girls and boys. It is said to be more common than the adolescent or young adult syndrome and is generally far less serious dermatologically and psychologically. An estimated 35 to 40 percent of patients with hair-pulling disorder chew or swallow the hair that they pull out at one time or another. Of this group, approximately onethird develop potentially hazardous bezoars—hairballs accumulating in the alimentary tract.

ETIOLOGY:

Although hair-pulling disorder is regarded as multidetermined, its onset has been linked to stressful situations in more than one-fourth of all cases. Disturbances in mother–child relationships, fear of being left alone, and recent object loss are often cited as critical factors contributing to the condition. Substance abuse may encourage development of the disorder. Depressive dynamics are often cited as predisposing factors, but no particular personality trait or disorder characterizes patients. Some see self-stimulation as the primary goal of hair pulling. Family members of hair-pulling disorder patients often have a history of tics, impulsecontrol disorders, and obsessive-compulsive symptoms, further supporting a possible genetic predisposition. One study looked at the neurobiology of hair-pulling disorder and found a smaller volume of the left putamen and left lenticulate areas. More recently, a study of the genetics of trichotillomania reported a relationship between a serotonin 2A (5-HT2A) receptor gene polymorphism (T102C) and trichotillomania. However, because these studies examined relatively few subjects, these findings need to be replicated in a larger sample to be able to determine the role of basal ganglia abnormalities and serotonin in the etiology of trichotillomania.

DIAGNOSIS AND CLINICAL FEATURES:

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) includes diagnostic criteria from hair-pulling disorder. Before engaging in the behavior, patients with hair-

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pulling disorder may experience an increasing sense of tension and achieve a sense of release or gratification from pulling out their hair. All areas of the body may be affected, most commonly the scalp. Other areas involved are eyebrows, eyelashes, and beard; trunk, armpits, and pubic area are less commonly involved.

Two types of hair pulling have been described. Focused pulling is the use of an intentional act to control unpleasant personal experiences, such as an urge, bodily sensation (e.g., itching or burning), or thought. In contrast, automatic pulling occurs outside the person's awareness and most often during sedentary activities. Most patients have a combination of these types of hair pulling. Hair loss is characterized by short, broken strands appearing together with long, normal hairs in the affected areas. No abnormalities of the skin or scalp are present. Hair pulling is not reported as being painful, although pruritus and tingling may occur in the involved area. Trichophagy, mouthing of the hair, may follow the hair plucking. Complications of trichophagy include trichobezoars, malnutrition, and intestinal obstruction. Patients usually deny the behavior and often try to hide the resultant alopecia. Head banging, nail biting, scratching, gnawing, excoriation, and other acts of self-mutilation may be present.

DIFFERENTIAL DIAGNOSIS:

Hair pulling may be a wholly benign condition or it may occur in the context of several mental disorders. The phenomenology of hair-pulling disorder and OCD overlap. As with OCD, hair-pulling disorder is often chronic and recognized by patients as undesirable. Unlike those with OCD, patients with hair-pulling disorder do not experience obsessive thoughts, and the compulsive activity is limited to one act, hair pulling. Patients with factitious disorder actively seek medical attention and the patient role and deliberately simulate illness toward these ends. Patients who malinger or who have factitious disorder may mutilate themselves to get medical attention, but they do not acknowledge the self-inflicted nature of the lesions. Patients with stereotypic movement disorder have stereotypical and rhythmic movements, and they usually do not seem distressed by their behavior. A biopsy may be necessary to distinguish hair-pulling disorder from alopecia areata and tine capitis.

COURSE AND PROGNOSIS:

The mean age at onset of hair-pulling disorder is in the early teens, most frequently before age 17, but onset has been reported much later in life. The course of the disorder is not well known; both chronic and remitting forms occur. An early onset (before age 6) tends to remit more readily and responds to suggestions, support, and behavioral strategies. Late onset (after age 13) is associated with an increased likelihood of chronicity and poorer prognosis than the early-onset form. About a third of persons presenting for treatment report a duration of 1 year or less, whereas in some cases, the disorder has persisted for more than two decades.

TREATMENT:

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No consensus exists on the best treatment modality for hair-pulling disorder. Treatment usually involves psychiatrists and dermatologists in a joint endeavor. Psychopharmalogical methods that have been used to treat psychodermatological disorders include topical steroids and hydroxyzine hydrochloride (Vistaril), an anxiolytic with antihistamine properties; antidepressants; and antipsychotics. Initial case reports showed efficacy of selective serotonin reuptake inhibitors (SSRIs) for hair-pulling disorder. Patients who respond poorly to SSRIs may improve with augmentation with pimozide (Orap), a dopamine receptor antagonist. Other medications that have been reported to have some efficacy for hair-pulling disorder include fluvoxamine (Luvox), citalopram (Celexa), venlafaxine (Effexor), naltrexone (ReVia), and lithium (Eskalith). A report of successful lithium treatment cited the possible effect of the drug on aggression, impulsivity, and mood instability as an explanation. In one study, patients taking naltrexone had a reduction in symptom severity. Case reports also indicate successful treatment with buspirone (BuSpar), clonazepam (Klonopin), and trazodone (Desyrel). Successful behavioral treatments, such as biofeedback, self-monitoring, desensitization, and habit reversal, have been reported, but most studies have been based on individual cases or a small series of cases with relatively short follow-up periods. Chronic hair-pulling disorder has been treated successfully with insight-oriented psychotherapy. Hypnotherapy has been mentioned as potentially effective in the treatment of dermatological disorders in which psychological factors may be involved; the skin has been shown to be susceptible to hypnotic suggestion.

5. **Excoriation (Skin-Picking) Disorder**

Excoriation or skin-picking disorder is characterized by the compulsive and repetitive picking of the skin. It can lead to severe tissue damage and result in the need for various dermatological treatments. Throughout history, skin-picking disorder has had many names: skin-picking syndrome, emotional excoriation, nervous scratching artifact, epidermotillomania, and para-artificial excoriation.

EPIDEMIOLOGY:

Skin-picking disorder has lifetime prevalence between 1 to 5 percent in the general population, about 12 percent in the adolescent psychiatric population, and occurs in 2 percent of patients with other dermatologic disorders. It is more prevalent in women than in men.

ETIOLOGY:

The cause of skin-picking is unknown, however, several theories have been postulated. Some theorists speculate that skin-picking behavior is a manifestation of repressed rage at authoritarian parents. These patients pick at their skin and perform other selfdestructive acts to assert themselves. Patients may pick as a means to relieve stress. For example, skin-picking has been associated with marital conflicts, passing of loved ones, and unwanted pregnancies. According to psychoanalytic theory, the skin is an erotic organ, and picking at the skin or scratching the skin leading to excoriations may be a source of erotic pleasure. In that sense it has been considered a masturbatory equivalent. Patients may be unaware of these affects presumed to be in the

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unconscious. Many patients begin picking at the onset of dermatological conditions such as acne and continue to pick after the condition has cleared. Abnormalities in serotonin, dopamine, and glutamate metabolism have been theorized to be an underlying neurochemical cause of the disorder, but further research is needed.

DIAGNOSIS: The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) it was called trichotillomania. It was also known as skin-picking syndrome. DSM-5 diagnostic criteria for skin-picking disorder requires recurrent skin-picking resulting in skin lesions and repeated attempts to decrease or stop picking. The skin-picking must cause clinically relevant distress or impairment in functioning. The skin-picking behavior cannot be attributed to another medical or mental condition and cannot be a result of a substance use disorder (e.g., cocaine or methamphetamine use).

CLINICAL FEATURES:

The face is the most common site of skin-picking (Fig. 10.5-1). Other common sites are legs, arms, torso, hands, cuticles, fingers, and scalp. Although most patients report having a primary picking area, many times they pick other areas of the body in order for the primary area to heal. In severe cases, skin-picking can result in physical disfigurement and medical consequences that require medical or surgical interventions (e.g., skin grafts or radiosurgery).

Patients may experience tension prior to picking and a relief and gratification after picking. Many report picking as a means to relieve stress, tension, and other negative feelings. In spite of the relief felt from picking, patients often feel guilty or embarrassed at their behavior. Up to 87 percent of patients report feeling embarrassed by the picking and 58 percent report avoiding social situations. Many patients use bandages, makeup, or clothing to hide their picking. Of skin-picking patients, 15 percent report suicidal ideation due to their behavior and about 12 percent have attempted suicide.

DIFFERENTIAL DIAGNOSIS:

The diagnosis of skin-picking disorder cannot be made if the behavior can be better accounted for by another medical or psychological condition. Many medical and dermatological conditions may result in urges to itch and pick at the skin. Conditions include eczema, psoriasis, diabetes, liver or kidney disease, Hodgkin's disease, polycythemia vera, or systemic lupus. Skin-picking can also be seen in Prader-Willi syndrome (97 percent). A thorough physical examination is crucial prior to psychiatric diagnosis. Skin-picking disorder is similar to OCD and it is associated with high rates of comorbid OCD. The disorders differ in a few ways. Skin-picking disorder is prevalent in females while OCD is equal between genders. The compulsions associated with OCD are usually driven by intrusive thoughts, while the compulsion to pick the skin is usually not. Although skin-picking generally decreases anxiety, it can also entice pleasure in the patient, which is rarely the case in OCD. Skin-picking in OCD patients is usually the result of obsessions about contamination or skin abnormalities. Skin-picking is commonly seen in body dysmorphic disorder. In one study, 45 percent of body dysmorphic patients report lifetime skin-picking disorder and 37 percent report having skin-picking disorder secondary to body dysmorphic disorder. The skin-picking in body dysmorphic disorder is

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primarily centered on removing or minimizing believed imperfection in the patient's appearance. Substance use disorders often co-occur with skin-picking disorder. Methamphetamine and cocaine use may result in the sensation that something is crawling on the body or under the skin (formication), which can result in skin-picking. In order to make the diagnosis of skin-picking disorder, however, skin-picking cannot be a physiological effect of substance use.

COURSE AND PROGNOSIS:

The onset of skin-picking disorder is either in early adulthood or between 30 and 45 years of age. Onset in children before age 10 years has also been seen. The mean age of onset is between 12 to 16 years of age. There may be a lag of time between onset and actual diagnosis. Because little is known about the disorder, many are unaware that it can be treated. Many times patients do not seek treatment until a severe dermatological or medical condition has developed. Typically, symptoms wax and wane over the course of the patient's life. Approximately 44 percent of women report that the amount of picking coincides with their menstrual cycle.

TREATMENT:

Skin-picking disorder is difficult to treat and there are few data on effective treatments. Most patients do not actively seek treatment due to embarrassment or because they believe their condition is untreatable. There is support for the use of selective serotonin reuptake inhibitors (SSRIs). Studies comparing fluoxetine (Prozac) against placebo has shown fluoxetine to be superior in reducing skin-picking. The opioid antagonist naltrexone (Revia) has proven to reduce the urge to pick, particularly in patients who experience pleasure from the behavior. Glutamatergic agents and lamotrigine (Lamictal) have also shown efficacy. Nonpharmacological treatments include habit reversal and brief cognitive-behavioral therapy (CBT). Effective therapy requires both psychological and somatic treatment. In some cases mechanical prevention of skin-picking by different protective measures may be of use in an effort to break the cycle. Psychotherapy at the same time deals with the underlying emotional factors.

Trauma- and Stressor-Related Disorders

1. Post-traumatic Stress Disorder and Acute Stress Disorder

Both posttraumatic stress disorder (PTSD) and acute stress disorder are marked by increased stress and anxiety following exposure to a traumatic or stressful event. Traumatic or stressful events may include being a witness to or being involved in a violent accident or crime, military combat, or assault, being kidnapped, being involved in a natural disaster, being diagnosed with a life-threatening illness, or experiencing systematic physical or sexual abuse. The person reacts to the experience with fear and helplessness, persistently relives the event, and tries to avoid being reminded of it. The event may be relived in dreams and waking thoughts (flashbacks). The stressors causing both acute stress disorder and PTSD are sufficiently overwhelming to affect almost

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everyone. They can arise from experiences in war, torture (discussed in detail below), natural catastrophes, assault, rape, and serious accidents, for example, in cars and in burning buildings. Persons reexperience the traumatic event in their dreams and their daily thoughts; they are determined to avoid anything that brings the event to mind and they undergo a numbing of responsiveness along with a state of hyperarousal. Other symptoms are depression, anxiety, and cognitive difficulties such as poor concentration.

EPIDEMIOLOGY:

The lifetime incidence of PTSD is estimated to be 9 to 15 percent and the lifetime prevalence of PTSD is estimated to be about 8 percent of the general population, although an additional 5 to 15 percent may experience subclinical forms of the disorder. The lifetime prevalence rate is 10 percent in women and 4 percent in men. According to the National Vietnam Veterans Readjustment Study (NVVRS), 30 percent of men develop full-blown PTSD after having served in the war and an additional 22.5 percent develop partial PTSD, falling just short of qualifying for the disorder. Among veterans of the Iraq and Afghanistan wars, 13 percent received the diagnosis of PTSD. Although PTSD can appear at any age, it is most prevalent in young adults, because they tend to be more exposed to precipitating situations. Children can also have the disorder (see Section 31.11b). Men and women differ in the types of traumas to which they are exposed. Historically, men's trauma was usually combat experience, and women's trauma was most commonly assault or rape. The disorder is most likely to occur in those who are single, divorced, widowed, socially withdrawn, or of low socioeconomic level, but anyone can be effected, no one is immune. The most important risk factors, however, for this disorder are the severity, duration, and proximity of a person's exposure to the actual trauma. A familial pattern seems to exist for this disorder, and first-degree biological relatives of persons with a history of depression have an increased risk for developing PTSD following a traumatic event.

ETIOLOGY:

Stressor By definition, a stressor is the prime causative factor in the development of PTSD. Not everyone experiences the disorder after a traumatic event, however. The stressor alone does not suffice to cause the disorder. Clinicians must also consider individual's preexisting biological and psychosocial factors and events that happened before and after the trauma. For example, a member of a group who lived through a disaster can sometimes better deal with trauma because others have also shared the experience. The stressor's subjective meaning to a person is also important. For example, survivors of a catastrophe may experience guilt feelings (survivor guilt) that can predispose to, or exacerbate, PTSD.

Psychodynamic Factors- The psychoanalytic model of the PTSD hypothesizes that the trauma has reactivated a previously quiescent, yet unresolved psychological conflict. The revival of the childhood trauma results in regression and the use of the defense mechanisms of repression, denial, reaction formation, and undoing. According to Freud, a splitting of consciousness occurs in patients who reported a history of childhood sexual trauma. A preexisting conflict might be symbolically

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reawakened by the new traumatic event. The ego relives and thereby tries to master and reduce the anxiety. Psychodynamic themes in PTSD are summarized in Table 11.1-2. Persons who suffer from alexithymia, the inability to identify or verbalize feeling states, are incapable of soothing themselves when under stress.

Biological Factors- The biological theories of PTSD have developed both from preclinical studies of animal models of stress and from measures of biological variables in clinical populations with the disorder. Many neurotransmitter systems have been implicated by both sets of data. Preclinical models of learned helplessness, kindling, and sensitization in animals have led to theories about norepinephrine, dopamine, endogenous opioids, and benzodiazepine receptors and the hypothalamic-pituitary-adrenal (HPA) axis. In clinical populations, data have supported hypotheses that the noradrenergic and endogenous opiate systems, as well as the HPA axis, are hyperactive in at least some patients with PTSD. Other major biological findings are increased activity and responsiveness of the autonomic nervous system, as evidenced by elevated heart rates and blood pressure readings and by abnormal sleep architecture (e.g., sleep fragmentation and increased sleep latency). Some researchers have suggested a similarity between PTSD and two other psychiatric disorders: major depressive disorder and panic disorder.

DIAGNOSIS:

The 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria for PTSD (Table 11.1-3) specify that the symptoms of intrusion, avoidance, alternations of mood and cognition, and hyperarousal must have lasted more than 1 month. The DSM-5 diagnosis of PTSD allows the physician to specify if the symptoms occur in preschool-aged children or with dissociative (depersonalization/derealization) symptoms. For patients whose symptoms have been present less than 1 month, the appropriate diagnosis may be acute stress disorder.

CLINICAL FEATURES:

Individuals with PTSD show symptoms in three domains: intrusion symptoms following the trauma, avoiding stimuli associated with the trauma, and experiencing symptoms of increased automatic arousal, such as an enhanced startle. Flashbacks, in which the individual may act and feel as if the trauma were reoccurring, represent a classic intrusion symptom. Other intrusion symptoms include distressing recollections or dreams and either physiological or psychological stress reactions to exposure to stimuli that are linked to the trauma. An individual must exhibit at least one intrusion symptom to meet the criteria for PTSD. Symptoms of avoidance associated with PTSD include efforts to avoid thoughts or activities related to the trauma, anhedonia, reduced capacity to remember events related to the trauma, blunted affect, feelings of detachment or derealization, and a sense of a foreshortened future. Symptoms of increased arousal include insomnia, irritability, hypervigilance, and exaggerated startle.

DIFFERENTIAL DIAGNOSIS:

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Because patients often exhibit complex reactions to trauma, the clinician must be careful to exclude other syndromes as well when evaluating patients presenting in the wake of trauma. It is particularly important to recognize potentially treatable medical contributors to posttraumatic symptomatology, especially head injury during the trauma. Medical contributors can usually be detected through a careful history and physical examination. Other organic considerations that can both cause and exacerbate the symptoms are epilepsy, alcohol-use disorders, and other substance-related disorders. Acute intoxication or withdrawal from some substances may also present a clinical picture that is difficult to distinguish from the disorder until the effects of the substance have worn off. Symptoms of PTSD can be difficult to distinguish from both panic disorder and generalized anxiety disorder, because all three syndromes are associated with prominent anxiety and autonomic arousal. Keys to correctly diagnosing PTSD involve a careful review of the time course relating the symptoms to a traumatic event. PTSD is also associated with re-experiencing and avoidance of a trauma, features typically not present in panic or generalized anxiety disorder. Major depression is also a frequent concomitant of PTSD. Although the two syndromes are not usually difficult to distinguish phenomenologically, it is important to note the presence of comorbid depression, because this can influence treatment of PTSD. PTSD must be differentiated from a series of related disorders that can exhibit phenomenological similarities, including borderline personality disorder, dissociative disorders, and factitious disorders. Borderline personality disorder can be difficult to distinguish from PTSD. The two disorders can coexist or even be causally related. Patients with dissociative disorders do not usually have the degree of avoidance behavior, the autonomic hyperarousal, or the history of trauma that patients with PTSD report.

COURSE AND PROGNOSIS:

PTSD usually develops some time after the trauma. The delay can be as short as 1 week or as long as 30 years. Symptoms can fluctuate over time and may be most intense during periods of stress. Untreated, about 30 percent of patients recover completely, 40 percent continue to have mild symptoms, 20 percent continue to have moderate symptoms, and 10 percent remain unchanged or become worse. After 1 year, about 50 percent of patients will recover. A good prognosis is predicted by rapid onset of the symptoms, short duration of the symptoms (less than 6 months), good premorbid functioning, strong social supports, and the absence of other psychiatric, medical, or substance-related disorders or other risk factors. In general, the very young and the very old have more difficulty with traumatic events than do those in midlife. For example, about 80 percent of young children who sustain a burn injury show symptoms of PTSD 1 or 2 years after the initial injury; only 30 percent of adults who suffer such an injury have symptoms of PTSD after 1 year. Presumably, young children do not yet have adequate coping mechanisms to deal with the physical and emotional insults of the trauma. Likewise, older persons are likely to have more rigid coping mechanisms than younger adults and to be less able to muster a flexible approach to dealing with the effects of trauma. Furthermore, the traumatic effects can be exacerbated by physical disabilities characteristic of late life, particularly disabilities of the nervous system and the cardiovascular system, such as reduced cerebral blood flow, failing vision, palpitations, and arrhythmias. Preexisting psychiatric disability, whether a personality disorder or a more serious condition, also increases the

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effects of particular stressors. PTSD that is comorbid with other disorders is often more severe and perhaps more chronic and may be difficult to treat. The availability of social supports may also influence the development, severity, and duration of PTSD. In general, patients who have a good network of social support are less likely to have the disorder and to experience it in its severe forms and are more likely to recover faster.

TREATMENT:

When a clinician is faced with a patient who has experienced a significant trauma, the major approaches are support, encouragement to discuss the event, and education about a variety of coping mechanisms (e.g., relaxation). In encouraging persons to talk about the event it is imperative that the clinician allow the person to proceed at his or her own pace. Some patients will not be willing to talk until well after the event has passed, and those wishes should be respected. To press a person who is reluctant to talk about a trauma into doing so is likely to increase rather than decrease the risk of developing PTSD. The use of sedatives and hypnotics can also be helpful in some cases. When a patient has experienced a traumatic event in the past and has now developed PTSD, the emphasis should be on education about the disorder and its treatment, both pharmacological and psychotherapeutic. The clinician should also work to destigmatize the notion of mental illness and PTSD. Additional support for the patient and the family can be obtained through local and national support groups for patients with PTSD.

2. Adjustment Disorders

The diagnostic category of adjustment disorders is widely used among clinicians in practice. Adjustment disorders are characterized by an emotional response to a stressful event. It is one of the few diagnostic entities in which an external stressful event is linked to the development of symptoms. Typically, the stressor involves financial issues, a medical illness, or relationship problem. The symptom complex that develops may involve anxious or depressive affect or may present with a disturbance of conduct. By definition, the symptoms must begin within 3 months of the stressor. A variety of subtypes of adjustment disorder are identified in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). These include adjustment disorder with depressed mood, mixed anxiety and depressed mood, disturbance of conduct, mixed disturbance of emotions and conduct, features of acute stress disorder or posttraumatic stress disorder (PTSD), bereavement, and unspecified type.

EPIDEMIOLOGY:

The prevalence of the disorder is estimated to be from 2 to 8 percent of the general population. Women are diagnosed with the disorder twice as often as men, and single women are generally overly represented as most at risk. In children and adolescents, boys and girls are equally diagnosed with adjustment disorders. The disorders can occur at any age but are most frequently diagnosed in adolescents. Among adolescents of either sex, common precipitating stresses are school problems, parental rejection and divorce, and substance abuse. Among adults, common precipitating stresses are marital problems, divorce, moving to a new environment, and financial problems. Adjustment

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disorders are one of the most common psychiatric diagnoses for disorders of patients hospitalized for medical and surgical problems. In one study, 5 percent of persons admitted to a hospital over a 3-year period were classified as having an adjustment disorder. Up to 50 percent of persons with specific medical problems or stressors have been diagnosed with adjustment disorders. Furthermore, 10 to 30 percent of mental health outpatients and up to 50 percent of general hospital inpatients referred for mental health consultations have been diagnosed with adjustment disorders.

ETIOLOGY:

By definition, an adjustment disorder is precipitated by one or more stressors. The severity of the stressor or stressors does not always predict the severity of the disorder; the stressor severity is a complex function of degree, quantity, duration, reversibility, environment, and personal context. For example, the loss of a parent is different for a child 10 years of age than for a person 40 years of age. Personality organization and cultural or group norms and values also contribute to the disproportionate responses to stressors. Stressors may be single, such as a divorce or the loss of a job, or multiple, such as the death of a person important to a patient, which coincides with the patient's own physical illness and loss of a job. Stressors may be recurrent, such as seasonal business difficulties, or continuous, such as chronic illness or poverty. A discordant intrafamilial relationship can produce an adjustment disorder that affects the entire family system, or the disorder may be limited to a patient who was perhaps the victim of a crime or who has a physical illness. Sometimes, adjustment disorders occur in a group or community setting, and the stressors affect several persons, as in a natural disaster or in racial, social, or religious persecution. Specific developmental stages, such as beginning school, leaving home, getting married, becoming a parent, failing to achieve occupational goals, having the last child leave home, and retiring, are often associated with adjustment disorders.

DIAGNOSIS AND CLINICAL FEATURES:

Although by definition adjustment disorders follow a stressor, the symptoms do not necessarily begin immediately. Up to 3 months may elapse between a stressor and the development of symptoms. Symptoms do not always subside as soon as the stressor ceases; if the stressor continues, the disorder may be chronic. The disorder can occur at any age, and its symptoms vary considerably, with depressive, anxious, and mixed features most common in adults. Physical symptoms, which are most common in children and the elderly, can occur in any age group. Manifestations may also include assaultive behavior and reckless driving, excessive drinking, defaulting on legal responsibilities, withdrawal, vegetative signs, insomnia, and suicidal behavior. The clinical presentations of adjustment disorder can vary widely. DSM-5 lists six adjustment disorders, including an unspecified category.

Adjustment Disorder with Depressed Mood-

In adjustment disorder with depressed mood, the predominant manifestations are depressed mood, tearfulness, and hopelessness. This type must be distinguished from major depressive disorder and

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uncomplicated bereavement. Adolescents with this type of adjustment disorder are at increased risk for major depressive disorder in young adulthood.

Adjustment Disorder with Anxiety-

Symptoms of anxiety, such as palpitations, jitteriness, and agitation, are present in adjustment disorder with anxiety, which must be differentiated from anxiety disorders.

Adjustment Disorder with Mixed Anxiety and Depressed Mood-

In adjustment disorder with mixed anxiety and depressed mood, patients exhibit features of both anxiety and depression that do not meet the criteria for an already established anxiety disorder or depressive disorder.

Adjustment Disorder with Disturbance of Conduct-

In adjustment disorder with disturbance of conduct, the predominant manifestation involves conduct in which the rights of others are violated or age-appropriate societal norms and rules are disregarded. Examples of behavior in this category are truancy, vandalism, reckless driving, and fighting. The category must be differentiated from conduct disorder and antisocial personality disorder.

Adjustment Disorder with Mixed Disturbance of Emotions and Conduct-

A combination of disturbances of emotions and of conduct sometimes occurs. Clinicians are encouraged to try to make one or the other diagnosis in the interest of clarity.

Adjustment Disorder Unspecified-

Adjustment disorder unspecified is a residual category for atypical maladaptive reactions to stress. Examples include inappropriate responses to the diagnosis of physical illness, such as massive denial, severe noncompliance with treatment, and social withdrawal, without significant depressed or anxious mood.

DIFFERENTIAL DIAGNOSIS:

Although uncomplicated bereavement often produces temporarily impaired social and occupational functioning, the person's dysfunction remains within the expectable bounds of a reaction to the loss of a loved one and, thus, is not considered adjustment disorder. See Section 34.1 for a further discussion of bereavement. Other disorders from which adjustment disorder must be differentiated include major depressive disorder, brief psychotic disorder, generalized anxiety disorder, somatic symptom disorder, substance-related disorder, conduct disorder, and PTSD. These diagnoses should be given precedence in all cases that meet their criteria, even in the presence of a stressor or group of stressors that served as a precipitant. Patients with an adjustment disorder are impaired in social or occupational functioning and show symptoms beyond the normal and expectable reaction to the stressor. Because no absolute criteria help to distinguish an adjustment disorder from another

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condition, clinical judgment is necessary. Some patients may meet the criteria for both an adjustment disorder and a personality disorder. If the adjustment disorder follows a physical illness, the clinician must make sure that the symptoms are not a continuation or another manifestation of the illness or its treatment.

Acute and Posttraumatic Stress Disorders-

The presence of a stressor is a requirement in the diagnosis of adjustment disorder, PTSD, and acute stress disorder. PTSD and acute stress disorder have the nature of the stressor better characterized and are accompanied by a defined constellation of affective and autonomic symptoms. In contrast, the stressor in adjustment disorder can be of any severity, with a wide range of possible symptoms. When the response to an extreme stressor does not meet the acute stress or posttraumatic disorder threshold, the adjustment disorder diagnosis would be appropriate. PTSD is discussed fully in Section 11.1.

COURSE AND PROGNOSIS:

With appropriate treatment, the overall prognosis of an adjustment disorder is generally favorable. Most patients return to their previous level of functioning within 3 months. Some persons (particularly adolescents) who receive a diagnosis of an adjustment disorder later have mood disorders or substance-related disorders. Adolescents usually require a longer time to recover than adults. Research over the past 5 years has disclosed a risk for suicide, especially in adolescent patients with adjustment disorder, not previously fully appreciated. A recent study of 119 patients with adjustment disorder indicated that 60 percent had documented suicide attempts in the hospital. Fifty percent had attempted suicide immediately prior to their hospital admission. Comorbid diagnoses of substance abuse and personality disorder contributed to the suicide risk profile. A study of the background, pathology, and treatment-related factors of suicidal adolescents found that those with adjustment disorder and suicidality were more likely to have made attempts (up to 25 percent), to exhibit psychomotor restlessness and dysphoric mood, to have experienced a suicide of another person as a stressor, to have poor psychosocial functioning upon treatment entry, and to have received prior psychiatric care. A 16-year-old high school senior experienced rejection in his first serious relationship. In the weeks after the end of the relationship, he began to exhibit dysphoric mood accompanied by anxiety and psychomotor agitation. He had received counseling in junior high school when his parents divorced and he began using alcohol and marijuana and had been suspended during his freshman year for fighting. A month after the breakup, he began to tell his parents that life was no longer worth living without his former girlfriend. Two months later his parents came home from work and found him hanging in the garage with a note stating he could not go on alone.

TREATMENT:

Psychotherapy Psychotherapy remains the treatment of choice for adjustment disorders. Group therapy can be particularly useful for patients who have had similar stresses—for example, a group of retired persons or patients having renal dialysis. Individual psychotherapy offers the opportunity

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to explore the meaning of the stressor to the patient so that earlier traumas can be worked through. After successful therapy, patients sometimes emerge from an adjustment disorder stronger than in the premorbid period, although no pathology was evident during that period. Because a stressor can be clearly delineated in adjustment disorders, it is often believed that psychotherapy is not indicated and that the disorder will remit spontaneously. This viewpoint, however, ignores the fact that many persons exposed to the same stressor experience different symptoms, and in adjustment disorders, the response is pathological. Psychotherapy can help persons adapt to stressors that are not reversible or time limited and can serve as a preventive intervention if the stressor does remit. Psychiatrists treating adjustment disorders must be particularly aware of problems of secondary gain. The illness role may be rewarding to some normally healthy persons who have had little experience with illness's capacity to free them from responsibility. Thus, patients can find therapists' attention, empathy, and understanding, which are necessary for success, rewarding in their own right, and therapists may thereby reinforce patients' symptoms. Such considerations must be weighed before intensive psychotherapy is begun; when a secondary gain has already been established, therapy is difficult. Patients with an adjustment disorder that includes a conduct disturbance may have difficulties with the law, authorities, or school. Psychiatrists should not attempt to rescue such patients from the consequences of their actions. Too often, such kindness only reinforces socially unacceptable means of tension reduction and hinders the acquisition of insight and subsequent emotional growth. In these cases, family therapy can help.

Pharmacotherapy-

No studies have assessed the efficacy of pharmacological interventions in individuals with adjustment disorder, but it may be reasonable to use medication to treat specific symptoms for a brief time. The judicious use of medications can help patients with adjustment disorders, but they should be prescribed for brief periods. Depending on the type of adjustment disorder, a patient may respond to an antianxiety agent or to an antidepressant. Patients with severe anxiety bordering on panic can benefit from anxiolytics such as diazepam (Valium), and those in withdrawn or inhibited states may be helped by a short course of psychostimulant medication. Antipsychotic drugs may be used if there are signs of decompensation or impending psychosis. Selective serotonin reuptake inhibitors have been found useful in treating symptoms of traumatic grief. Recently, there has been an increase in antidepressant use to augment psychotherapy in patients with adjustment disorders. Pharmacological intervention in this population is most often used, however, to augment psychosocial strategies rather than serving as the primary modality.

Dissociative Disorders

1. DISSOCIATIVE IDENTITY DISORDER

Dissociative identity disorder, previously called multiple personality disorder, has been the most extensively researched of all the dissociative disorders. It is characterized by the presence of two or

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more distinct identities or personality states. The identities or personality states, sometimes called alters, self-states, alter identities, or parts, among other terms, differ from one another in that each presents as having its own pattern of perceiving, relating to, and thinking about the environment and self, in short, its own personality. It is the paradigmatic dissociative psychopathology in that the symptoms of all the other dissociative disorders are commonly found in patients with dissociative identity disorder: amnesia, fugue, depersonalization, derealization, and similar symptoms.

EPIDEMIOLOGY:

Few systematic epidemiological data exist for dissociative identity disorder. Clinical studies report female to male ratios between 5 to 1 and 9 to 1 for diagnosed cases.

ETIOLOGY:

Dissociative identity disorder is strongly linked to severe experiences of early childhood trauma, usually maltreatment. The rates of reported severe childhood trauma for child and adult patients with dissociative identity disorder range from 85 to 97 percent of cases. Physical and sexual abuse are the most frequently reported sources of childhood trauma. The contribution of genetic factors is only now being systematically assessed, but preliminary studies have not found evidence of a significant genetic contribution.

DIAGNOSIS AND CLINICAL FEATURES:

The key feature in diagnosing this disorder is the presence of two or more distinct personality states. There are many other signs and symptoms, however, that define the disorder, and because of great diversity, this make the diagnosis difficult

DIFFERENTIAL DIAGNOSIS:

Anxiety, psychotic, affective, ptsd, personality, neurocognitive, neurological and seizure, somatic, factitious, other dissociative, deep trance phenomenon.

COURSE AND PROGNOSIS:

Little is known about the natural history of untreated dissociative identity disorder. Some individuals with untreated dissociative identity disorder are thought to continue involvement in abusive relationships or violent subcultures, or both, that may result in the traumatization of their children, with the potential for additional family transmission of the disorder. Many authorities believe that some percentage of patients with undiagnosed or untreated dissociative identity disorder die by suicide or as a result of their risk-taking behaviors. Prognosis is poorer in patients with comorbid organic mental disorders, psychotic disorders (not dissociative identity disorder pseudopsychosis), and severe medical illnesses. Refractory substance abuse and eating disorders also suggest a poorer prognosis. Other factors that usually indicate a poorer outcome include significant antisocial personality features, current criminal activity, ongoing perpetration of abuse,

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and current victimization, with refusal to leave abusive relationships. Repeated adult traumas with recurrent episodes of acute stress disorder may severely complicate the clinical course.

TREATMENT:

-Psychotherapy

-Cognitive Therapy

-Hypnosis

-Psychopharmacological Interventions

-Electroconvulsive Therapy

2. DEPERSONALIZATION/DEREALIZATION DISORDER

Depersonalization is defined as the persistent or recurrent feeling of detachment or estrangement from one's self. The individual may report feeling like an automaton or watching himself or herself in a movie (Fig. 12-1). Derealization is somewhat related and refers to feelings of unreality or of being detached from one's environment. The patient may describe his or her perception of the outside world as lacking lucidity and emotional coloring, as though dreaming or dead.

EPIDEMIOLOGY:

Transient experiences of depersonalization and derealization are extremely common in normal and clinical populations. They are the third most commonly reported psychiatric symptoms, after depression and anxiety. One survey found a 1-year prevalence of 19 percent in the general population. It is common in seizure patients and migraine sufferers; they can also occur with use of psychedelic drugs, especially marijuana, lysergic acid diethylamide (LSD), and mescaline, and less frequently as a side effect of some medications, such as anticholinergic agents. They have been described after certain types of meditation, deep hypnosis, extended mirror or crystal gazing, and sensory deprivation experiences. They are also common after mild to moderate head injury, wherein little or no loss of consciousness occurs, but they are significantly less likely if unconsciousness lasts for more than 30 minutes. They are also common after life-threatening experiences, with or without serious bodily injury. Depersonalization is found two to four times more in women than in men.

ETIOLOGY:

Psychodynamic-

Traditional psychodynamic formulations have emphasized the disintegration of the ego or have viewed depersonalization as an affective response in defense of the ego. These explanations stress the role of overwhelming painful experiences or conflictual impulses as triggering events.

Traumatic Stress-

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A substantial proportion, typically one third to one half, of patients in clinical depersonalization case series report histories of significant trauma. Several studies of accident victims find as many as 60 percent of those with a lifethreatening experience report at least transient depersonalization during the event or immediately thereafter. Military training studies find that symptoms of depersonalization and derealization are commonly evoked by stress and fatigue and are inversely related to performance.

Neurobiological Theories-

The association of depersonalization with migraines and marijuana, its generally favorable response to selective serotonin reuptake inhibitors (SSRIs), and the increase in depersonalization symptoms seen with the depletion of L-tryptophan, a serotonin precursor, point to serotoninergic involvement. Depersonalization is the primary dissociative symptom elicited by the drug-challenge studies described in the section on neurobiological theories of dissociation. These studies strongly implicate the N-methyl-D-aspartate (NMDA) subtype of the glutamate receptor as central to the genesis of depersonalization symptoms.

DIAGNOSIS AND CLINICAL FEATURES:

A number of distinct components comprise the experience of depersonalization, including a sense of (1) bodily changes, (2) duality of self as observer and actor, (3) being cut off from others, and (4) being cut off from one's own emotions. Patients experiencing depersonalization often have great difficulty expressing what they are feeling. Trying to express their subjective suffering with banal phrases, such as "I feel dead," "Nothing seems real," or "I'm standing outside of myself," depersonalized patients may not adequately convey to the examiner the distress they experience. While complaining bitterly about how this is ruining their life, they may nonetheless appear remarkably undistressed.

DIFFERENTIAL DIAGNOSIS:

The variety of conditions associated with depersonalization complicate the differential diagnosis of depersonalization disorder. Depersonalization can result from a medical condition or neurological condition, intoxication or withdrawal from illicit drugs, as a side effect of medications, or can be associated with panic attacks, phobias, PTSD, or acute stress disorder, schizophrenia, or another dissociative disorder. A thorough medical and neurological evaluation is essential, including standard laboratory studies, an EEG, and any indicated drug screens. Drug-related depersonalization is typically transient, but persistent depersonalization can follow an episode of intoxication with a variety of substances, including marijuana, cocaine, and other psychostimulants. A range of neurological conditions, including seizure disorders, brain tumors, postconcussive syndrome, metabolic abnormalities, migraine, vertigo, and Ménière's disease, have been reported as causes. Depersonalization caused by organic conditions tends to be primarily sensory without the elaborated descriptions and personalized meanings common to psychiatric etiologies.

COURSE AND PROGNOSIS:

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Depersonalization after traumatic experiences or intoxication commonly remits spontaneously after removal from the traumatic circumstances or ending of the episode of intoxication. Depersonalization accompanying mood, psychotic, or other anxiety disorders commonly remits with definitive treatment of these conditions. Depersonalization disorder itself may have an episodic, relapsing and remitting, or chronic course. Many patients with chronic depersonalization may have a course characterized by severe impairment in occupational, social, and personal functioning. Mean age of onset is thought to be in late adolescence or early adulthood in most cases.

TREATMENT:

Clinicians working with patients with depersonalization/derealization disorder often find them to be a singularly clinically refractory group. Some systematic evidence indicates that SSRI antidepressants, such as fluoxetine (Prozac), may be helpful to patients with depersonalization disorder. Two recent, double-blind, placebo-controlled studies, however, found no efficacy for fluvoxamine (Luvox) and lamotrigine (Lamictal), respectively, for depersonalization disorder. Some patients with depersonalization disorder respond at best sporadically and partially to the usual groups of psychiatric medications, singly or in combination: antidepressants, mood stabilizers, typical and atypical neuroleptics, anticonvulsants, and so forth. Many different types of psychotherapy have been used to treat depersonalization disorder: psychodynamic, cognitive, cognitive-behavioral, hypnotherapeutic, and supportive. Many such patients do not have a robust response to these specific types of standard psychotherapy. Stress management strategies, distraction techniques, reduction of sensory stimulation, relaxation training, and physical exercise may be somewhat helpful in some patients.

3. DISSOCIATIVE AMNESIA

The DSM-5 diagnostic criteria for dissociative amnesia are listed in Table 12-1. The main feature of dissociative amnesia is an inability to recall important personal information, usually of a traumatic or stressful nature, that is too extensive to be explained by normal forgetfulness. And, as mentioned above, the disorder does not result from the direct physiological effects of a substance or a neurological or other general medical condition.

Types:

- Localized- inability to recall events related to a circumscribed period of time.
- Selective- ability to remember some, but not all, of the events occurring during circumscribed period of time.
- Generalized- failure to recall one's entire life.
- Continuous- failure to recall successive events as they occur.
- Systematized- failure to recall a category of information, such as all memories relating to one's family or to a particular person.

EPIDEMIOLOGY:

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Dissociative amnesia has been reported in a range of approximately 2 to 6 percent of the general population. No known difference is seen in incidence between men and women. Cases generally begin to be reported in late adolescence and adulthood. Dissociative amnesia can be especially difficult to assess in preadolescent children because of their more limited ability to describe subjective experience.

ETIOLOGY:

In many cases of acute dissociative amnesia, the psychosocial environment out of which the amnesia develops is massively conflictual, with the patient experiencing intolerable emotions of shame, guilt, despair, rage, and desperation. These usually result from conflicts over unacceptable urges or impulses, such as intense sexual, suicidal, or violent compulsions. Traumatic experiences such as physical or sexual abuse can induce the disorder. In some cases the trauma is caused by a betrayal by a trusted, needed other (betrayal trauma). This betrayal is thought to influence the way in which the event is processed and remembered.

DIAGNOSIS AND CLINICAL FEATURES:

Classic Presentation-

The classic disorder is an overt, florid, dramatic clinical disturbance that frequently results in the patient being brought quickly to medical attention, specifically for symptoms related to the dissociative disorder. It is frequently found in those who have experienced extreme acute trauma. It also commonly develops, however, in the context of profound intrapsychic conflict or emotional stress. Patients may present with intercurrent somatoform or conversion symptoms, alterations in consciousness, depersonalization, derealization, trance states, spontaneous age regression, and even ongoing anterograde dissociative amnesia. Depression and suicidal ideation are reported in many cases. No single personality profile or antecedent history is consistently reported in these patients, although a prior personal or family history of somatoform or dissociative symptoms has been shown to predispose individuals to develop acute amnesia during traumatic circumstances. Many of these patients have histories of prior adult or childhood abuse or trauma. In wartime cases, as in other forms of combat-related posttraumatic disorders, the most important variable in the development of dissociative symptoms, however, appears to be the intensity of combat. Table 12-3 presents the mental status evaluation of dissociative amnesia.

Non-classic Presentation-

These patients frequently come to treatment for a variety of symptoms, such as depression or mood swings, substance abuse, sleep disturbances, somatoform symptoms, anxiety and panic, suicidal or self-mutilating impulses and acts, violent outbursts, eating problems, and interpersonal problems. Selfmutilation and violent behavior in these patients may also be accompanied by amnesia. Amnesia may also occur for flashbacks or behavioral re-experiencing episodes related to trauma.

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COURSE AND PROGNOSIS:

Little is known about the clinical course of dissociative amnesia. Acute dissociative amnesia frequently spontaneously resolves once the person is removed to safety from traumatic or overwhelming circumstances. At the other extreme, some patients do develop chronic forms of generalized, continuous, or severe localized amnesia and are profoundly disabled and require high levels of social support, such as nursing home placement or intensive family caretaking. Clinicians should try to restore patients' lost memories to consciousness as soon as possible; otherwise, the repressed memory may form a nucleus in the unconscious mind around which future amnestic episodes may develop.

TREATMENT:

-Cognitive Therapy

- Hypnosis

- Somatic Therapies

-Group Psychotherapy

4. DISSOCIATIVE FUGUE

Dissociative fugue was deleted as a major diagnostic category in DSM-5 and is now diagnosed on a subtype (specifier) of dissociative amnesia. Dissociative fugue can be seen in patients with both dissociative amnesia and dissociative identity disorder. The disorder remains a distinct diagnosis in the International Statistical Classification of Diseases and Related Health Problems tenth edition (ICD-10) and is discussed as a discrete entity in Synopsis because of its clinical relevance. Dissociative fugue is described as sudden, unexpected travel away from home or one's customary place of daily activities, with inability to recall some or all of one's past. This is accompanied by confusion about personal identity or even the assumption of a new identity. The disturbance is not due to the direct physiological effects of a substance or a general medical condition. The symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

ETIOLOGY:

Traumatic circumstances (i.e., combat, rape, recurrent childhood sexual abuse, massive social dislocations, natural disasters), leading to an altered state of consciousness dominated by a wish to flee, are the underlying cause of most fugue episodes. In some cases a similar antecedent history is seen, although a psychological trauma is not present at the onset of the fugue episode. In these cases, instead of, or in addition to, external dangers or traumas, the patients are usually struggling with extreme emotions or impulses (i.e., overwhelming fear, guilt, shame, or intense incestuous, sexual, suicidal, or violent urges) that are in conflict with the patient's conscience or ego ideals.

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EPIDEMIOLOGY:

The disorder is thought to be more common during natural disasters, wartime, or times of major social dislocation and violence, although no systematic data exist on this point. No adequate data exist to demonstrate a gender bias to this disorder; however, most cases describe men, primarily in the military. Dissociative fugue is usually described in adults.

DIAGNOSIS AND CLINICAL FEATURES:

Dissociative fugues have been described to last from minutes to months. Some patients report multiple fugues. In most cases in which this was described, a more chronic dissociative disorder, such as dissociative identity disorder, was not ruled out. In some extremely severe cases of PTSD, nightmares may be terminated by a waking fugue in which the patient runs to another part of the house or runs outside. Children or adolescents may be more limited than adults in their ability to travel. Thus, fugues in this population may be brief and involve only short distances.

DIFFERENTIAL DIAGNOSIS:

Individuals with dissociative amnesia may engage in confused wandering during an amnesia episode. In dissociative fugue, however, there is purposeful travel away from the individual's home or customary place of daily activities, usually with the individual preoccupied by a single idea that is accompanied by a wish to run away. Patients with dissociative identity disorder may have symptoms of dissociative fugue, usually recurrently throughout their lives. Patients with dissociative identity disorder have multiple forms of complex amnesias and, usually, multiple alter identities that develop, starting in childhood. In complex partial seizures, patients have been noted to exhibit wandering or semipurposeful behavior, or both, during seizures or in postictal states, for which subsequent amnesia occurs. Seizure patients in an epileptic fugue often exhibit abnormal behavior, however, including confusion, perseveration, and abnormal or repetitive movements. Other features of seizures are typically reported in the clinical history, such as an aura, motor abnormalities, stereotyped behavior, perceptual alterations, incontinence, and a postictal state. Serial or telemetric EEGs, or both, usually show abnormalities associated with behavioral pathology. Wandering behavior during a variety of general medical conditions, toxic and substance-related disorders, delirium, dementia, and organic amnestic syndromes could theoretically be confused with dissociative fugue. In most cases, however, the somatic, toxic, neurological, or substance-related disorder can be ruled in by the history, physical examination, laboratory tests, or toxicological and drug screening. Use of alcohol or substances may be involved in precipitating an episode of dissociative fugue. Wandering and purposeful travel can occur during the manic phase of bipolar disorder or schizoaffective disorder. Patients who are manic may not recall behavior that occurred in the euthymic or depressed state and vice versa. In purposeful travel owing to mania, however, the patient is usually preoccupied with grandiose ideas and often calls attention to himself or herself because of inappropriate behavior. Assumption of an alternate identity does not occur. Similarly, peripatetic behavior can occur in some patients with schizophrenia. Memory for events during wandering episodes in such patients may be difficult to ascertain owing to the patient's thought disorder. Patients with dissociative fugue, however, do not demonstrate a psychotic thought disorder or

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other symptoms of psychosis. Malingering of dissociative fugue can occur in individuals who are attempting to flee a situation involving legal, financial, or personal difficulties, as well as in soldiers who are attempting to avoid combat or unpleasant military duties. No test, battery of tests, or set of procedures exist that invariably distinguish true dissociative symptoms from those that are malingered. Malingering of dissociative symptoms, such as reports of amnesia for purposeful travel during an episode of antisocial behavior, can be maintained even during hypnotic or pharmacologically facilitated interviews. Many malingerers confess spontaneously or when confronted. In the forensic context, the examiner should always carefully consider the diagnosis of malingering when fugue is claimed.

COURSE AND PROGNOSIS:

Most fugues are relatively brief, lasting from hours to days. Most individuals appear to recover, although refractory dissociative amnesia may persist in rare cases. Some studies have described recurrent fugues in most individuals presenting with an episode of dissociative fugue. No systematic modern data exist that attempt to differentiate dissociative fugue from dissociative identity disorder with recurrent fugues.

TREATMENT:

Dissociative fugue is usually treated with an eclectic, psychodynamically oriented psychotherapy that focuses on helping the patient recover memory for identity and recent experience. Hypnotherapy and pharmacologically facilitated interviews are frequently necessary adjunctive techniques to assist with memory recovery. Patients may need medical treatment for injuries sustained during the fugue as well as food and sleep. Clinicians should be prepared for the emergence of suicidal ideation or self-destructive ideas and impulses as the traumatic or stressful prefugue circumstances are revealed. Psychiatric hospitalization may be indicated if the patient is an outpatient. Family, sexual, occupational, or legal problems that were part of the original matrix that generated the fugue episode may be substantially exacerbated by the time the patient's original identity and life situation are detected. Thus, family treatment and social service interventions may be necessary to help resolve such complex difficulties. When dissociative fugue involves assumption of a new identity, it is useful to conceptualize this entity as psychologically vital to protecting the person. Traumatic experiences, memories, cognitions, identifications, emotions, strivings, self-perceptions, or a combination of these have become so conflicting and, yet, so peremptory that the person can resolve them only by embodying them in an alter identity. The therapeutic goal in such cases is neither suppression of the new identity nor fascinated explication of all its attributes. As in dissociative identity disorder, the clinician should appreciate the importance of the psychodynamic information contained within the alter personality state and the intensity of the psychological forces that necessitated its creation. In these cases, the most desirable therapeutic outcome is fusion of the identities, with the person working through and integrating the memories of the experiences that precipitated the fugue.

Schizophrenia Spectrum and Other Psychotic Disorders

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1. Schizophrenia

Signs and symptoms are variable and include changes in perception, emotion, cognition, thinking, and behavior. The disorder usually begins before age 25 years, persists throughout life, and affects persons of all social classes. Diagnosis of schizophrenia is based entirely on the psychiatric history and mental status examination. There is no laboratory test for schizophrenia. Two major figures in psychiatry and neurology who studied the disorder were Emil Kraepelin (1856–1926) and Eugene Bleuler (1857–1939).

EPIDEMIOLOGY:

In the United States, the lifetime prevalence of schizophrenia is about 1 percent, which means that about one person in 100 will develop schizophrenia during their lifetime. The Epidemiologic Catchment Area study sponsored by the National Institute of Mental Health reported a lifetime prevalence of 0.6 to 1.9 percent. In the United States, about 0.05 percent of the total population is treated for schizophrenia in any single year, and only about half of all patients with schizophrenia obtain treatment, despite the severity of the disorder.

Gender and Age-

Schizophrenia is equally prevalent in men and women. The two genders differ, however, in the onset and course of illness. Onset is earlier in men than in women. More than half of all male schizophrenia patients, but only one-third of all female schizophrenia patients, are first admitted to a psychiatric hospital before age 25 years. The peak ages of onset are 10 to 25 years for men and 25 to 35 years for women. Unlike men, women display a bimodal age distribution, with a second peak occurring in middle age. Approximately 3 to 10 percent of women with schizophrenia present with disease onset after age 40 years. About 90 percent of patients in treatment for schizophrenia are between 15 and 55 years old.

ETIOLOGY:

The monozygotic twin data clearly demonstrate the fact that individuals who are genetically vulnerable to schizophrenia do not inevitably develop schizophrenia; other factors (e.g., environment) must be involved in determining a schizophrenia outcome.

Genetic Factors-

The modes of genetic transmission in schizophrenia are unknown, but several genes appear to make a contribution to schizophrenia vulnerability. Linkage and association genetic studies have provided strong evidence for nine linkage sites: 1q, 5q, 6p, 6q, 8p, 10p, 13q, 15q, and 22q. Further analyses of these chromosomal sites have led to the identification of specific candidate genes, and the best current candidates are α-7 nicotinic receptor, DISC 1, GRM 3, COMT, NRG 1, RGS 4, and G 72. Recently, mutations of the genes dystrobrevin (DTNBP1) and neureglin 1 have been found to be associated with negative features of schizophrenia.

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Biochemical Factors-

Dopamine Hypothesis- schizophrenia results from too much dopaminergic activity. The theory evolved from two observations. First, the efficacy and the potency of many antipsychotic drugs (i.e., the dopamine receptor antagonists [DRAs]) are correlated with their ability to act as antagonists of the dopamine type 2 (D2) receptor. Second, drugs that increase dopaminergic activity, notably cocaine and amphetamine, are psychotomimetic.

Serotonin- Current hypotheses posit serotonin excess as a cause of both positive and negative symptoms in schizophrenia. The robust serotonin antagonist activity of clozapine and other second-generation antipsychotics coupled with the effectiveness of clozapine to decrease positive symptoms in chronic patients has contributed to the validity of this proposition.

Norepinephrine- Anhedonia—the impaired capacity for emotional gratification and the decreased ability to experience pleasure—has long been noted to be a prominent feature of schizophrenia. A selective neuronal degeneration within the norepinephrine reward neural system could account for this aspect of schizophrenic symptomatology. However, biochemical and pharmacological data bearing on this proposal are inconclusive.

GABA- The inhibitory amino acid neurotransmitter γ-aminobutyric acid (GABA) has been implicated in the pathophysiology of schizophrenia based on the finding that some patients with schizophrenia have a loss of GABAergic neurons in the hippocampus. GABA has a regulatory effect on dopamine activity, and the loss of inhibitory GABAergic neurons could lead to the hyperactivity of dopaminergic neurons.

Neuropeptides- Neuropeptides, such as substance P and neurotensin, are localized with the catecholamine and indolamine neurotransmitters and influence the action of these neurotransmitters. Alteration in neuropeptide mechanisms could facilitate, inhibit, or otherwise alter the pattern of firing these neuronal systems.

Glutamate- Glutamate has been implicated because ingestion of phencyclidine, a glutamate antagonist, produces an acute syndrome similar to schizophrenia. The hypotheses proposed about glutamate include those of hyperactivity, hypoactivity, and glutamate-induced neurotoxicity.

Acetylcholine and Nicotine- Postmortem studies in schizophrenia have demonstrated decreased muscarinic and nicotinic receptors in the caudate-putamen, hippocampus, and selected regions of the prefrontal cortex. These receptors play a role in the regulation of neurotransmitter systems involved in cognition, which is impaired in schizophrenia.

Neuropathology-

Researchers had made significant strides in revealing a potential neuropathological basis for schizophrenia, primarily in the limbic system and the basal ganglia, including neuropathological or neurochemical abnormalities in the cerebral cortex, the thalamus, and the brainstem. The loss of brain volume widely reported in schizophrenic brains appears to result from reduced density of the

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axons, dendrites, and synapses that mediate associative functions of the brain. Computed tomography (CT) scans of patients with schizophrenia have consistently shown lateral and third ventricular enlargement and some reduction in cortical volume. There is a reduced symmetry in several brain areas in schizophrenia, including the temporal, frontal, and occipital lobes. Because of its role in controlling emotions, the limbic system has been hypothesized to be involved in the pathophysiology of schizophrenia. Decrease in the size of the region, including the amygdala, the hippocampus, and the parahippocampal gyrus. The hippocampus is not only smaller in size in schizophrenia but is also functionally abnormal as indicated by disturbances in glutamate transmission. Anatomical abnormalities in the prefrontal cortex. Some studies of the thalamus show evidence of volume shrinkage or neuronal loss, in particular subnuclei. The medial dorsal nucleus of the thalamus, which has reciprocal connections with the prefrontal cortex, has been reported to contain a reduced number of neurons. The total number of neurons, oligodendrocytes, and astrocytes is reduced by 30 to 45 percent in schizophrenia patients. many patients with schizophrenia show odd movements, even in the absence of medication-induced movement disorders (e.g., tardive dyskinesia). The odd movements can include an awkward gait, facial grimacing, and stereotypies. Because the basal ganglia and cerebellum are involved in the control of movement, disease in these areas is implicated in the pathophysiology of schizophrenia. Second, the movement disorders involving the basal ganglia (e.g., Huntington's disease, Parkinson's disease) are the ones most commonly associated with psychosis.

PSYCHOSOCIAL AND PSYCHOANALYTIC THEORIES-

If schizophrenia is a disease of the brain, it is likely to parallel diseases of other organs (e.g., myocardial infarctions, diabetes) whose courses are affected by psychosocial stress. The disorder affects individual patients, each of whom has a unique psychological makeup. Sigmund Freud postulated that schizophrenia resulted from developmental fixations early in life. These fixations produce defects in ego development, and he postulated that such defects contributed to the symptoms of schizophrenia. Ego disintegration in schizophrenia represents a return to the time when the ego was not yet developed or had just begun to be established. Because the ego affects the interpretation of reality and the control of inner drives, such as sex and aggression, these ego functions are impaired. Thus, intrapsychic conflict arising from the early fixations and the ego defect, which may have resulted from poor early object relations, fuel the psychotic symptoms.

DIAGNOSIS:

The DSM-5 diagnostic criteria include course specifiers (i.e., prognosis) that offer clinicians several options and describe actual clinical situations (Table 7.1-1). The presence of hallucinations or delusions is not necessary for a diagnosis of schizophrenia; the patient's disorder is diagnosed as schizophrenia when the patient exhibits two of the symptoms listed in symptoms 1 through 5 of Criterion A in Table 7.1-1 (e.g., disorganized speech). Criterion B requires that impaired functioning, although not deteriorations, be present during the active phase of the illness. Symptoms must persist for at least 6 months, and a diagnosis of schizoaffective disorder or mood disorder must be absent.

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Subtypes-

Five subtypes of schizophrenia have been described based predominantly on clinical presentation: paranoid, disorganized, catatonic, undifferentiated, and residual. DSM-5 no longer uses these subtypes but they are listed in the 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10).

Paranoid Type- The paranoid type of schizophrenia is characterized by preoccupation with one or more delusions or frequent auditory hallucinations. Classically, the paranoid type of schizophrenia is characterized mainly by the presence of delusions of persecution or grandeur. Patients with the paranoid type of schizophrenia show less regression of their mental faculties, emotional responses, and behavior than do patients with other types of schizophrenia. Patients with paranoid schizophrenia are typically tense, suspicious, guarded, reserved, and sometimes hostile or aggressive, but they can occasionally conduct themselves adequately in social situations. Their intelligence in areas not invaded by their psychosis tends to remain intact.

Disorganized Type- The disorganized type of schizophrenia is characterized by a marked regression to primitive, disinhibited, and unorganized behavior and by the absence of symptoms that meet the criteria for the catatonic type. Their thought disorder is pronounced, and their contact with reality is poor. Their personal appearance is disheveled, and their social behavior and their emotional responses are inappropriate. They often burst into laughter without any apparent reason. Incongruous grinning and grimacing are common in these patients, whose behavior is best described as silly or fatuous.

Catatonic Type- The classic feature of the catatonic type is a marked disturbance in motor function; this disturbance may involve stupor, negativism, rigidity, excitement, or posturing. Sometimes the patient shows a rapid alteration between extremes of excitement and stupor. Associated features include stereotypies, mannerisms, and waxy flexibility. Mutism is particularly common.

Undifferentiated Type- Frequently, patients who clearly have schizophrenia cannot be easily fit into one type or another. These patients are classified as having schizophrenia of the undifferentiated type.

Residual Type- The residual type of schizophrenia is characterized by continuing evidence of the schizophrenic disturbance in the absence of a complete set of active symptoms or of sufficient symptoms to meet the diagnosis of another type of schizophrenia. Emotional blunting, social withdrawal, eccentric behavior, illogical thinking, and mild loosening of associations commonly appear in the residual type. When delusions or hallucinations occur, they are neither prominent nor accompanied by strong affect.

CLINICAL FEATURES:

In theoretical formulations of the course of schizophrenia, premorbid signs and symptoms appear before the prodromal phase of the illness. The differentiation implies that premorbid signs and

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symptoms exist before the disease process evidences itself and that the prodromal signs and symptoms are parts of the evolving disorder. In the typical, but not invariable, premorbid history of schizophrenia, patients had schizoid or schizotypal personalities characterized as quiet, passive, and introverted; as children, they had few friends. Preschizophrenic adolescents may have no close friends and no dates and may avoid team sports. They may enjoy watching movies and television, listening to music, or playing computer games to the exclusion of social activities. Some adolescent patients may show a sudden onset of obsessive-compulsive behavior as part of the prodromal picture. The validity of the prodromal signs and symptoms, almost invariably recognized after the diagnosis of schizophrenia has been made, is uncertain; after schizophrenia is diagnosed, the retrospective remembrance of early signs and symptoms is affected. Nevertheless, although the first hospitalization is often believed to mark the beginning of the disorder, signs and symptoms have often been present for months or even years. The signs may have started with complaints about somatic symptoms, such as headache, back and muscle pain, weakness, and digestive problems. The initial diagnosis may be malingering, chronic fatigue syndrome, or somatization disorder. Family and friends may eventually notice that the person has changed and is no longer functioning well in occupational, social, and personal activities. During this stage, a patient may begin to develop an interest in abstract ideas, philosophy, and the occult or religious questions (Fig. 7.1-5). Additional prodromal signs and symptoms can include markedly peculiar behavior, abnormal affect, unusual speech, bizarre ideas, and strange perceptual experiences.

DIFFERENTIAL DIAGNOSIS:

Secondary Psychotic Disorders- A wide range of nonpsychiatric medical conditions and a variety of substances can induce symptoms of psychosis and catatonia (Table 7.1-3). The most appropriate diagnosis for such psychosis or catatonia is psychotic disorder due to a general medical condition, catatonic disorder due to a general medical condition, or substance-induced psychotic disorder.

Other Psychotic Disorders- The psychotic symptoms of schizophrenia can be identical with those of schizophreniform disorder, brief psychotic disorder, schizoaffective disorder, and delusional disorders. Schizophreniform disorder differs from schizophrenia in that the symptoms have a duration of at least 1 month but less than 6 months. Brief psychotic disorder is the appropriate diagnosis when the symptoms have lasted at least 1 day but less than 1 month and when the patient has not returned to the premorbid state of functioning within that time. There may also be a precipitating traumatic event. When a manic or depressive syndrome develops concurrently with the major symptoms of schizophrenia, schizoaffective disorder is the appropriate diagnosis. Nonbizarre delusions present for at least 1 month without other symptoms of schizophrenia or a mood disorder warrant the diagnosis of delusional disorder.

Mood Disorders- A patient with a major depressive episode may present with delusions and hallucinations, whether the patient has unipolar or bipolar mood disorder. Delusions seen with psychotic depression are typically mood congruent and involve themes such as guilt, self-depreciation, deserved punishment, and incurable illnesses. In mood disorders, psychotic symptoms resolve completely with the resolution of depression. A depressive episode that is this severe may

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also result in loss of functioning, decline in self-care, and social isolation, but these are secondary to the depressive symptoms and should not be confused with the negative symptoms of schizophrenia. A full-blown manic episode often presents with delusions and sometimes hallucinations. Delusions in mania are most often mood congruent and typically involve grandiose themes. The flight of ideas seen in mania may, at times, be confused with the thought disorder of schizophrenia. Special attention during mental status examination of a patient with a flight of ideas is required to note whether the associative links between topics are conserved, although the conversation is difficult for the observer to follow because of the patient's accelerated rate of thinking.

Personality Disorders- Various personality disorders may have some features of schizophrenia. Schizotypal, schizoid, and borderline personality disorders are the personality disorders with the most similar symptoms. Severe obsessive-compulsive personality disorder may mask an underlying schizophrenic process. Personality disorders, unlike schizophrenia, have mild symptoms and a history of occurring throughout a patient's life; they also lack an identifiable date of onset.

Malingering and Factitious Disorders- For a patient who imitates the symptoms of schizophrenia but does not actually have the disorder, either malingering or factitious disorder may be an appropriate diagnosis. Persons have faked schizophrenic symptoms and have been admitted into and treated at psychiatric hospitals. The condition of patients who are completely in control of their symptom production may qualify for a diagnosis of malingering; such patients usually have some obvious financial or legal reason to want to be considered mentally ill. The condition of patients who are less in control of their falsification of psychotic symptoms may qualify for a diagnosis of factitious disorder. Some patients with schizophrenia, however, may falsely complain of an exacerbation of psychotic symptoms to obtain increased assistance benefits or to gain admission to a hospital.

COURSE AND PROGNOSIS:

Course-

A premorbid pattern of symptoms may be the first evidence of illness, although the importance of the symptoms is usually recognized only retrospectively. Characteristically, the symptoms begin in adolescence and are followed by the development of prodromal symptoms in days to a few months. Social or environmental changes, such as going away to college, using a substance, or a relative's death, may precipitate the disturbing symptoms, and the prodromal syndrome may last a year or more before the onset of overt psychotic symptoms. The classic course of schizophrenia is one of exacerbations and remissions. After the first psychotic episode, a patient gradually recovers and may then function relatively normally for a long time. Patients usually relapse, however, and the pattern of illness during the first 5 years after the diagnosis generally indicates the patient's course. Further deterioration in the patient's baseline functioning follows each relapse of the psychosis. This failure to return to baseline functioning after each relapse is the major distinction between schizophrenia and the mood disorders. Sometimes a clinically observable postpsychotic depression follows a psychotic episode, and the schizophrenia patient's vulnerability to stress is usually lifelong. Positive symptoms tend to become less severe with time, but the socially debilitating negative or deficit

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symptoms may increase in severity. Although about one-third of all schizophrenia patients have some marginal or integrated social existence, most have lives characterized by aimlessness; inactivity; frequent hospitalizations; and, in urban settings, homelessness and poverty.

Prognosis-

Several studies have shown that over the 5- to 10-year period after the first psychiatric hospitalization for schizophrenia, only about 10 to 20 percent of patients can be described as having a good outcome. More than 50 percent of patients can be described as having a poor outcome, with repeated hospitalizations, exacerbations of symptoms, episodes of major mood disorders, and suicide attempts. Despite these glum figures, schizophrenia does not always run a deteriorating course, and several factors have been associated with a good prognosis.

Reported remission rates range from 10 to 60 percent, and a reasonable estimate is that 20 to 30 percent of all schizophrenia patients are able to lead somewhat normal lives. About 20 to 30 percent of patients continue to experience moderate symptoms, and 40 to 60 percent of patients remain significantly impaired by their disorder for their entire lives. Patients with schizophrenia do much poorer than patients with mood disorders, although 20 to 25 percent of mood disorder patients are also severely disturbed at long-term follow-up.

TREATMENT:

Although antipsychotic medications are the mainstay of the treatment for schizophrenia, research has found that psychosocial interventions, including psychotherapy, can augment the clinical improvement. Just as pharmacological agents are used to treat presumed chemical imbalances, nonpharmacological strategies must treat nonbiological issues. The complexity of schizophrenia usually renders any single therapeutic approach inadequate to deal with the multifaceted disorder. Psychosocial modalities should be integrated into the drug treatment regimen and should support it. Patients with schizophrenia benefit more from the combined use of antipsychotic drugs and psychosocial treatment than from either treatment used alone.

2. Schizoaffective Disorder

Schizoaffective disorder has features of both schizophrenia and mood disorders. In current diagnostic systems, patients can receive the diagnosis of schizoaffective disorder if they fit into one of the following six categories: (1) patients with schizophrenia who have mood symptoms, (2) patients with mood disorder who have symptoms of schizophrenia, (3) patients with both mood disorder and schizophrenia, (4) patients with a third psychosis unrelated to schizophrenia and mood disorder, (5) patients whose disorder is on a continuum between schizophrenia and mood disorder, and (6) patients with some combination of the above.

EPIDEMIOLOGY:

The lifetime prevalence of schizoaffective disorder is less than 1 percent, possibly in the range of 0.5 to 0.8 percent. These figures, however, are estimates; various studies of schizoaffective disorder

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have used varying diagnostic criteria. In clinical practice, a preliminary diagnosis of schizoaffective disorder is frequently used when a clinician is uncertain of the diagnosis.

Gender and Age Differences-

Sex differences in the rates of schizoaffective disorder in clinical samples generally parallel sex differences seen in mood disorders, with approximately equal numbers of men and women who have the bipolar subtype and are more than twofold female to male predominance among individuals with the depressed subtype of schizoaffective disorder. The depressive type of schizoaffective disorder may be more common in older persons than in younger persons, and the bipolar type may be more common in young adults than in older adults. The age of onset for women is later than that for men, as in schizophrenia. Men with schizoaffective disorder are likely to exhibit antisocial behavior and to have a markedly flat or inappropriate affect.

ETIOLOGY:

The cause of schizoaffective disorder is unknown. The disorder may be a type of schizophrenia, a type of mood disorder, or the simultaneous expression of each. Schizoaffective disorder may also be a distinct third type of psychosis, one that is unrelated to either schizophrenia or a mood disorder. The most likely possibility is that schizoaffective disorder is a heterogeneous group of disorders encompassing all of these possibilities. Studies designed to explore the etiology have examined family histories, biological markers, short-term treatment responses, and long-term outcomes. Most studies have considered patients with schizoaffective disorder to be a homogeneous group, but recent studies have examined the bipolar and depressive types of schizoaffective disorder separately. Although much of the family and genetic research in schizoaffective disorder is based on the premise that schizophrenia and the mood disorders are completely separate entities, some data indicate that they may be genetically related. Studies of the disrupted in schizophrenia 1 (DISC1) gene, located on chromosome 1q42, suggest its possible involvement in schizoaffective disorder as well as schizophrenia and bipolar disorder. As a group, patients with schizoaffective disorder have a better prognosis than patients with schizophrenia and a worse prognosis than patients with mood disorders. Also, as a group, patients with schizoaffective disorder tend to have a nondeteriorating course and respond better to lithium than do patients with schizophrenia.

DIAGNOSIS AND CLINICAL FEATURES:

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria for schizoaffective disorder are provided in Table 7.2-1. The clinician must accurately diagnose the affective illness, making sure it meets the criteria of either a manic or a depressive episode but also determining the exact length of each episode (not always easy or even possible). The length of each episode is critical for two reasons. First, to meet the Criterion B (psychotic symptoms in the absence of a major mood episode [depressive or manic]), it is important to know when the affective episode ends and the psychosis continues. Second, to meet Criterion C, the length of all mood episodes must be combined and compared with the total length of the illness. If the mood component is present for the majority (>50 percent) of the total illness, then that criterion is met. As with most psychiatric

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diagnoses, schizoaffective disorder should not be used if the symptoms are caused by substance abuse or a secondary medical condition.

DIFFERENTIAL DIAGNOSIS:

The psychiatric differential diagnosis includes all the possibilities usually considered for mood disorders and for schizophrenia. In any differential diagnosis of psychotic disorders, a complete medical workup should be performed to rule out organic causes for the symptoms. A history of substance use (with or without positive results on a toxicology screening test) may indicate a substance-induced disorder. Preexisting medical conditions, their treatment, or both can cause psychotic and mood disorders. Any suspicion of a neurological abnormality warrants consideration of a brain scan to rule out anatomical pathology and an electroencephalogram to determine any possible seizure disorders (e.g., temporal lobe epilepsy). Psychotic disorder caused by seizure disorder is more common than that seen in the general population. It tends to be characterized by paranoia, hallucinations, and ideas of reference. Patients with epilepsy with psychosis are believed to have a better level of function than patients with schizophrenic spectrum disorders. Better control of the seizures can reduce the psychosis.

COURSE AND PROGNOSIS:

Considering the uncertainty and evolving diagnosis of schizoaffective disorder, it is difficult to determine the long-term course and prognosis. Given the definition of the diagnosis, patients with schizoaffective disorder might be expected to have a course similar to an episodic mood disorder, a chronic schizophrenic course, or some intermediate outcome. It has been presumed that an increasing presence of schizophrenic symptoms predicted a worse prognosis. After 1 year, patients with schizoaffective disorder had different outcomes, depending on whether their predominant symptoms were affective (better prognosis) or schizophrenic (worse prognosis). One study that followed patients diagnosed with schizoaffective disorder for 8 years found that the outcomes of these patients more closely resembled schizophrenia than a mood disorder with psychotic features.

TREATMENT:

Mood stabilizers are a mainstay of treatment for bipolar disorders and would be expected to be important in the treatment of patients with schizoaffective disorder. One study that compared lithium with carbamazepine (Tegretol) found that carbamazepine was superior for schizoaffective disorder, depressive type, but found no difference in the two agents for the bipolar type. In practice, however, these medications are used extensively alone, in combination with each other, or with an antipsychotic agent. In manic episodes, patients who are schizoaffective should be treated aggressively with dosages of a mood stabilizer in the middle to high therapeutic blood concentration range. As the patient enters maintenance phase, the dosage can be reduced to a low to middle range to avoid adverse effects and potential effects on organ systems (e.g., thyroid and kidney) and to improve ease of use and compliance. Laboratory monitoring of plasma drug concentrations and periodic screening of thyroid, kidney, and hematological functioning should be performed. By definition, many patients who are schizoaffective have major depressive episodes. Treatment with

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antidepressants mirrors treatment of bipolar depression. Care should be taken not to precipitate a cycle of rapid switches from depression to mania with the antidepressant. The choice of antidepressant should take into account previous antidepressant successes or failures. Selective serotonin reuptake inhibitors (e.g., fluoxetine [Prozac] and sertraline [Zoloft]) are often used as first-line agents because they have less effect on cardiac status and have a favorable overdose profile. Agitated or insomniac patients, however, may benefit from a tricyclic drug. As in all cases of intractable mania, the use of ECT should be considered. As mentioned, antipsychotic agents are important in the treatment of the psychotic symptoms of schizoaffective disorder.

3. Schizophreniform Disorder

The concept of schizophreniform disorder was introduced in 1939 by Gabriel Langfeldt (1895–1983) to describe a condition with a sudden onset and benign course associated with mood symptoms and clouding of consciousness. The symptoms of schizophreniform are similar to those of schizophrenia; however, with schizophreniform disorder, the symptoms last for at least 1 month but less than 6 months. In contrast, for a patient to meet the diagnostic criteria for schizophrenia, the symptoms must have been present for at least 6 months. Patients with schizophreniform disorder return to their baseline level of functioning after the disorder has resolved.

EPIDEMIOLOGY:

Little is known about the incidence, prevalence, and sex ratio of schizophreniform disorder. The disorder is most common in adolescents and young adults and is less than half as common as schizophrenia. A fivefold greater rate of schizophreniform disorder has been found in men than in women. A 1-year prevalence rate of 0.09 percent and a lifetime prevalence rate of 0.11 percent have been reported. Several studies have shown that the relatives of patients with schizophreniform disorder are at high risk of having other psychiatric disorders, but the distribution of the disorders differs from the distribution seen in the relatives of patients with schizophrenia and bipolar disorders. Specifically, the relatives of patients with schizophreniform disorders are more likely to have mood disorders than are the relatives of patients with schizophrenia. In addition, the relatives of patients with schizophreniform disorder are more likely to have a diagnosis of a psychotic mood disorder than are the relatives of patients with bipolar disorders.

ETIOLOGY:

The cause of schizophreniform disorder is not known. As Langfeldt noted in 1939, patients with this diagnostic label are likely to be heterogeneous. In general, whereas some patients have a disorder similar to schizophrenia, others have a disorder similar to a mood disorder. Because of the generally good outcome, the disorder probably has similarities to the episodic nature of mood disorders. Some data, however, indicate a close relation to schizophrenia. In support of the relation to mood disorders, several studies have shown that patients with schizophreniform disorder, as a group, have more affective symptoms (especially mania) and a better outcome than patients with schizophrenia. Also, the increased occurrence of mood disorders in the relatives of patients with schizophreniform disorder indicates a relation to mood disorders. Thus, the biological and epidemiological data are

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most consistent with the hypothesis that the current diagnostic category defines a group of patients, some of whom have a disorder similar to schizophrenia; others have a disorder resembling a mood disorder.

DIAGNOSTIC AND CLINICAL FEATURES:

The DSM-5 criteria for schizophreniform disorder are listed in Table 7.3-1. Schizophreniform disorder is an acute psychotic disorder that has a rapid onset and lacks a long prodromal phase. Although many patients with schizophreniform disorder may experience functional impairment at the time of an episode, they are unlikely to report a progressive decline in social and occupational functioning. The initial symptom profile is the same as that of schizophrenia in that two or more psychotic symptoms (hallucinations, delusions, disorganized speech and behavior, or negative symptoms) must be present. Schneiderian first-rank symptoms are frequently observed. Also an increased likelihood is found of emotional turmoil and confusion, the presence of which may indicate a good prognosis. Although negative symptoms may be present, they are relatively uncommon in schizophreniform disorder and are considered poor prognostic features. In a small series of first-admission patients with schizophreniform, one-fourth had moderate to severe negative symptoms. Almost all were initially categorized as having “schizophreniform disorder without good prognostic features,” and 2 years later, 73 percent were rediagnosed with schizophrenia compared with 38 percent of those with “good prognostic features.” By definition, patients with schizophreniform disorder return to their baseline state within 6 months. In some instances, the illness is episodic, with more than one episode occurring after long periods of full remission. If the combined duration of symptomatology exceeds 6 months, however, then schizophrenia should be considered.

DIFFERENTIAL DIAGNOSIS:

It is important to first differentiate schizophreniform disorder from psychoses that can arise from medical conditions. This is accomplished by taking a detailed history and physical examination and, when indicated, performing laboratory tests or imaging studies. A detailed history of medication use, including over-the-counter medications and herbal products, is essential because many therapeutic agents can also produce an acute psychosis. Although it is not always possible to distinguish substance-induced psychosis from other psychotic disorders cross-sectionally, a rapid onset of psychotic symptoms in a patient with a significant substance history should raise the suspicion of a substanceinduced psychosis. A detailed substance use history and toxicological screen are also important for treatment planning in an individual with a new onset of psychosis. The duration of psychotic symptoms is one factor that distinguishes schizophreniform disorder from other syndromes. Schizophrenia is diagnosed if the duration of the prodromal, active, and residual phases lasts for more than 6 months; symptoms that occur for less than 1 month indicate a brief psychotic disorder. In general, a diagnosis of brief psychotic disorder does not require that a major stressor be present. Distinguishing mood disorders with psychotic features from schizophreniform disorder is sometimes difficult. Furthermore, schizophreniform disorder and schizophrenia can be highly comorbid with mood and anxiety disorders. Additional confounds are that mood symptoms,

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such as loss of interest and pleasure, may be difficult to distinguish from negative symptoms, avolition, and anhedonia. Some mood symptoms may also be present during the early course of schizophrenia. A thorough longitudinal history is important in elucidating the diagnosis because the presence of psychotic symptoms exclusively during periods of mood disturbance is an indication of a primary mood disorder.

COURSE AND PROGNOSIS:

The course of schizophreniform disorder, for the most part, is defined in the criteria. It is a psychotic illness lasting more than 1 month and less than 6 months. The real issue is what happens to persons with this illness over time. Most estimates of progression to schizophrenia range between 60 and 80 percent. What happens to the other 20 to 40 percent is currently not known. Some will have a second or third episode during which they will deteriorate into a more chronic condition of schizophrenia. A few, however, may have only this single episode and then continue on with their lives, which is clearly the outcome desired by all clinicians and family members, although it is probably a rare occurrence and should not be held out as likely.

TREATMENT:

Hospitalization, which is often necessary in treating patients with schizophreniform disorder, allows effective assessment, treatment, and supervision of a patient's behavior. The psychotic symptoms can usually be treated by a 3- to 6-month course of antipsychotic drugs (e.g., risperidone). Several studies have shown that patients with schizophreniform disorder respond to antipsychotic treatment much more rapidly than patients with schizophrenia. In one study, about 75 percent of patients with schizophreniform disorder and only 20 percent of the patients with schizophrenia responded to antipsychotic medications within 8 days. A trial of lithium (Eskalith), carbamazepine (Tegretol), or valproate (Depakene) may be warranted for treatment and prophylaxis if a patient has a recurrent episode. Psychotherapy is usually necessary to help patients integrate the psychotic experience into their understanding of their own minds and lives. ECT may be indicated for some patients, especially those with marked catatonic or depressed features. Finally, most patients with schizophreniform disorder progress to full-blown schizophrenia despite treatment. In those cases, a course of management consistent with a chronic illness must be formulated.

4. Delusional Disorder and Shared Psychotic Disorder

Delusions are false fixed beliefs not in keeping with the culture. They are among the most interesting of psychiatric symptoms because of the great variety of false beliefs that can be held by so many people and because they are so difficult to treat. The diagnosis of delusional disorder is made when a person exhibits nonbizarre delusions of at least 1 month's duration that cannot be attributed to other psychiatric disorders. Nonbizarre means that the delusions must be about situations that can occur in real life, such as being followed, infected, loved at a distance, and so on; that is, they usually have to do with phenomena that, although not real, are nonetheless possible. Several types of delusions may be present and the predominant type is specified when the diagnosis is made.

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EPIDEMIOLOGY:

An accurate assessment of the epidemiology of delusional disorder is hampered by the relative rareness of the disorder, as well as by its changing definitions in recent history. Moreover, delusional disorder may be underreported because delusional patients rarely seek psychiatric help unless forced to do so by their families or by the courts. Even with these limitations, however, the literature does support the contention that delusional disorder, although uncommon, has a relatively steady rate. The prevalence of delusional disorder in the United States is currently estimated to be 0.2 to 0.3 percent. Thus, delusional disorder is much rarer than schizophrenia, which has a prevalence of about 1 percent, and the mood disorders, which have a prevalence of about 5 percent. The annual incidence of delusional disorder is one to three new cases per 100,000 persons. The mean age of onset is about 40 years, but the range for age of onset runs from 18 years of age to the 90s. A slight preponderance of female patients exists. Men are more likely to develop paranoid delusions than women, who are more likely to develop delusions of erotomania. Many patients are married and employed, but some association is seen with recent immigration and low socioeconomic status.

ETIOLOGY:

As with all major psychiatric disorders, the cause of delusional disorder is unknown. Moreover, patients currently classified as having delusional disorder probably have a heterogeneous group of conditions with delusions as the predominant symptom. The central concept about the cause of delusional disorder is its distinctness from schizophrenia and the mood disorders. Delusional disorder is much rarer than either schizophrenia or mood disorders, with a later onset than schizophrenia and a much less pronounced female predominance than the mood disorders. The most convincing data come from family studies that report an increased prevalence of delusional disorder and related personality traits (e.g., suspiciousness, jealousy, and secretiveness) in the relatives of delusional disorder probands. Family studies have reported neither an increased incidence of schizophrenia and mood disorders in the families of delusional disorder probands nor an increased incidence of delusional disorder in the families of probands with schizophrenia. Long-term follow-up of patients with delusional disorder indicates that the diagnosis of delusional disorder is relatively stable, with fewer than one-fourth of the patients eventually being reclassified as having schizophrenia and fewer than 10 percent of patients eventually being reclassified as having a mood disorder. These data indicate that delusional disorder is not simply an early stage in the development of one or both of these two more common disorders.

Biological Factors-

A wide range of nonpsychiatric medical conditions and substances, including clear-cut biological factors, can cause delusions, but not everyone with a brain tumor, for example, has delusions. Unique, and not yet understood, factors in a patient’s brain and personality are likely to be relevant to the specific pathophysiology of delusional disorder. The neurological conditions most commonly associated with delusions affect the limbic system and the basal ganglia. Patients whose delusions

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are caused by neurological diseases and who show no intellectual impairment tend to have complex delusions similar to those in patients with delusional disorder. Conversely, patients with neurological disorder with intellectual impairments often have simple delusions unlike those in patients with delusional disorder. Thus, delusional disorder may involve the limbic system or basal ganglia in patients who have intact cerebral cortical functioning. Delusional disorder can arise as a normal response to abnormal experiences in the environment, the peripheral nervous system, or the central nervous system (CNS). Thus, if patients have erroneous sensory experiences of being followed (e.g., hearing footsteps), they may come to believe that they are actually being followed. This hypothesis hinges on the presence of hallucinatory-like experiences that need to be explained. The presence of such hallucinatory experiences in delusional disorder has not been proved.

Psychodynamic Factors-

Practitioners have a strong clinical impression that many patients with delusional disorder are socially isolated and have attained less than expected levels of achievement. Specific psychodynamic theories about the cause and the evolution of delusional symptoms involve suppositions regarding hypersensitive persons and specific ego mechanisms, which are reaction formation, projection, and denial.

DIAGNOSIS AND CLINICAL FEATURES:

TYPES-

Persecutory Type- Patients with this subtype are convinced that they are being persecuted or harmed. The persecutory beliefs are often associated with querulousness, irritability, and anger, and the individual who acts out his or her anger may at times be assaultive or even homicidal.

Jealous Type- Delusional disorder with delusions of infidelity has been called conjugal paranoia when it is limited to the delusion that a spouse has been unfaithful. The delusion usually affects men, often those with no prior psychiatric illness. It may appear suddenly and serve to explain a host of present and past events involving the spouse's behavior. The condition is difficult to treat and may diminish only on separation, divorce, or death of the spouse.

Erotomanic Type- the patient has the delusional conviction that another person, usually of higher status, is in love with him or her. Such patients also tend to be solitary, withdrawn, dependent, and sexually inhibited as well as to have poor levels of social or occupational functioning. The following operational criteria for the diagnosis of erotomania have been suggested: (1) a delusional conviction of amorous communication, (2) object of much higher rank, (3) object being the first to fall in love, (4) object being the first to make advances, (5) sudden onset (within a 7-day period), (6) object remains unchanged, (7) patient rationalizes paradoxical behavior of the object, (8) chronic course, and (9) absence of hallucinations. Patients with erotomania frequently show certain characteristics: They are generally unattractive women in low-level jobs who lead withdrawn, lonely lives; they are single and have few sexual contacts. They select secret lovers who differ substantially from them. They exhibit what has been called paradoxical conduct, the delusional phenomenon of interpreting

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all denials of love, no matter how clear, as secret affirmations of love. The course may be chronic, recurrent, or brief.

Somatic Type- Delusional disorder with somatic delusions has been called monosymptomatic hypochondriacal psychosis. The delusion is fixed, unarguable, and presented intensely because the patient is totally convinced of the physical nature of the disorder. In contrast, persons with hypochondriasis often admit that their fear of illness is largely groundless. The content of the somatic delusion can vary widely from case to case. The three main types are (1) delusions of infestation (including parasitosis); (2) delusions of dysmorphophobia, such as of misshapenness, personal ugliness, or exaggerated size of body parts (this category seems closest to that of body dysmorphic disorder); and (3) delusions of foul body odors or halitosis. The onset of symptoms with the somatic type of delusional disorder may be gradual or sudden. In most patients, the illness is unremitting, although the delusion severity may fluctuate. Hyperalertness and high anxiety also characterize patients with this subtype.

Grandiose Type- Delusions of grandeur (megalomania) have been noted for years. They were first described by Kraepelin.

Mixed Type- The category mixed type applies to patients with two or more delusional themes. This diagnosis should be reserved for cases in which no single delusional type predominates.

Unspecified Type- The category unspecified type is reserved for cases in which the predominant delusion cannot be subtyped within the previous categories. A possible example is certain delusions of misidentification, for example, Capgras syndrome. The delusion in Capgras syndrome is the belief that a familiar person has been replaced by an impostor.

DIFFERENTIAL DIAGNOSIS:

Toxic-metabolic conditions and disorders affecting the limbic system and basal ganglia are most often associated with the emergence of delusional beliefs. In Huntington’s disease and in individuals with idiopathic basal ganglia calcifications, for example, more than 50 percent of patients demonstrated delusions at some point in their illness. Capgras syndrome has been observed in a number of medical disorders, including CNS lesions, vitamin B12 deficiency, hepatic encephalopathy, diabetes, and hypothyroidism. Delusions of infestation, lycanthropy (i.e., the false belief that the patient is an animal, often a wolf or “werewolf”), heutoscopy (i.e., the false belief that one has a double), and erotomania have been reported in small numbers of patients with epilepsy, CNS lesions, or toxic-metabolic disorders.

Delirium and dementia should be considered in the differential diagnosis of a patient with delusions. Delirium can be differentiated by the presence of a fluctuating level of consciousness or impaired cognitive abilities. Delusions early in the course of a dementing illness, as in dementia of the Alzheimer’s type, can give the appearance of a delusional disorder; however, neuropsychological testing usually detects cognitive impairment. Although alcohol abuse is an associated feature for patients with delusional disorder, delusional disorder should be distinguished from alcohol-induced

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psychotic disorder with hallucinations. Intoxication with sympathomimetics (including amphetamine), marijuana, or L-dopa is likely to result in delusional symptoms.

Other Disorders-

The psychiatric differential diagnosis for delusional disorder includes malingering and factitious disorder with predominantly psychological signs and symptoms. The nonfactitious disorders in the differential diagnosis are schizophrenia, mood disorders, obsessive-compulsive disorder, somatoform disorders, and paranoid personality disorder. Delusional disorder is distinguished from schizophrenia by the absence of other schizophrenic symptoms and by the nonbizarre quality of the delusions; patients with delusional disorder also lack the impaired functioning seen in schizophrenia. The somatic type of delusional disorder may resemble a depressive disorder or a somatoform disorder. The somatic type of delusional disorder is differentiated from depressive disorders by the absence of other signs of depression and the lack of a pervasive quality to the depression. Delusional disorder can be differentiated from somatoform disorders by the degree to which the somatic belief is held by the patient. Patients with somatoform disorders allow for the possibility that their disorder does not exist, but patients with delusional disorder do not doubt its reality. Separating paranoid personality disorder from delusional disorder requires the sometimes difficult clinical distinction between extreme suspiciousness and frank delusion. In general, if clinicians doubt that a symptom is a delusion, the diagnosis of delusional disorder should not be made.

COURSE AND PROGNOSIS:

Some clinicians and some research data indicate that an identifiable psychosocial stressor often accompanies the onset of delusional disorder. The nature of the stressor, in fact, may warrant some suspicion or concern. Examples of such stressors are recent immigration, social conflict with family members or friends, and social isolation. A sudden onset is generally thought to be more common than an insidious onset. Some clinicians believe that a person with delusional disorder is likely to have below-average intelligence and that the premorbid personality of such a person is likely to be extroverted, dominant, and hypersensitive. The person's initial suspicions or concerns gradually become elaborate, consume much of the person's attention, and finally become delusional. Persons may begin quarreling with coworkers; may seek protection from the Federal Bureau of Investigation (FBI) or the police; or may begin visiting many medical or surgical physicians to seek consultations, lawyers about suits, or police about delusional suspicions. As mentioned, delusional disorder is considered a fairly stable diagnosis. About 50 percent of patients have recovered at long-term follow-up, 20 percent show decreased symptoms, and 30 percent exhibit no change. The following factors correlate with a good prognosis: high levels of occupational, social, and functional adjustments; female sex; onset before age 30 years; sudden onset; short duration of illness; and the presence of precipitating factors. Although reliable data are limited, patients with persecutory, somatic, and erotic delusions are thought to have a better prognosis than patients with grandiose and jealous delusions.

TREATMENT:

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Delusional disorder was generally regarded as resistant to treatment, and interventions often focused on managing the morbidity of the disorder by reducing the impact of the delusion on the patient's (and family's) life. In recent years, however, the outlook has become less pessimistic or restricted in planning effective treatment. The goals of treatment are to establish the diagnosis, to decide on appropriate interventions, and to manage complications (Table 7.4-4). The success of these goals depends on an effective and therapeutic doctor–patient relationship, which is far from easy to establish. The patients do not complain about psychiatric symptoms and often enter treatment against their will; even psychiatrists may be drawn into their delusional nets.

5. BRIEF PSYCHOTIC DISORDER

Brief psychotic disorder is defined as a psychotic condition that involves the sudden onset of psychotic symptoms, which lasts 1 day or more but less than 1 month. Remission is full, and the individual returns to the premorbid level of functioning. Brief psychotic disorder is an acute and transient psychotic syndrome.

EPIDEMIOLOGY:

The exact incidence and prevalence of brief psychotic disorder is not known, but it is generally considered uncommon. The disorder occurs more often among younger patients (20s and 30s) than among older patients. Brief psychotic disorder is more common in women than in men. Such epidemiological patterns are sharply distinct from those of schizophrenia. Some clinicians indicate that the disorder may be seen most frequently in patients from low socioeconomic classes and in those who have experienced disasters or major cultural changes (e.g., immigrants). The age of onset in industrialized settings may be higher than in developing countries. Persons who have gone through major psychosocial stressors may be at greater risk for subsequent brief psychotic disorder.

ETIOLOGY:

The cause of brief psychotic disorder is unknown. Patients who have a personality disorder may have a biological or psychological vulnerability for the development of psychotic symptoms, particularly those with borderline, schizoid, schizotypal, or paranoid qualities. Some patients with brief psychotic disorder have a history of schizophrenia or mood disorders in their families, but this finding is nonconclusive. Psychodynamic formulations have emphasized the presence of inadequate coping mechanisms and the possibility of secondary gain for patients with psychotic symptoms. Additional psychodynamic theories suggest that the psychotic symptoms are a defense against a prohibited fantasy, the fulfillment of an unattained wish, or an escape from a stressful psychosocial situation.

DIAGNOSIS:

A diagnosis of brief psychotic disorder is appropriate when psychotic symptoms last at least 1 day but less than 1 month and are not associated with a mood disorder, a substance-related disorder, or a psychotic disorder caused by a general medical condition. There are three subtypes of brief

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psychotic disorder: (1) the presence of a stressor, (2) the absence of a stressor, and (3) a postpartum onset. As with other acutely ill psychiatric patients, the history necessary to make the diagnosis may not be obtainable solely from the patient. Although psychotic symptoms may be obvious, information about prodromal symptoms, previous episodes of a mood disorder, and a recent history of ingestion of a psychotomimetic substance may not be available from the clinical interview alone. In addition, clinicians may not be able to obtain accurate information about the presence or absence of precipitating stressors. Such information is usually best and most accurately obtained from a relative or a friend.

CLINICAL FEATURES:

The symptoms of brief psychotic disorder always include at least one major symptom of psychosis, such as hallucinations, delusions, and disorganized thoughts, usually with an abrupt onset, but do not always include the entire symptom pattern seen in schizophrenia. Some clinicians have observed that labile mood, confusion, and impaired attention may be more common at the onset of brief psychotic disorder than at the onset of eventually chronic psychotic disorders. Characteristic symptoms in brief psychotic disorder include emotional volatility, strange or bizarre behavior, screaming or muteness, and impaired memory of recent events. Some of the symptoms suggest a diagnosis of delirium and warrant a medical workup, especially to rule out adverse reactions to drugs. Scandinavian and other European literature describes several characteristic symptom patterns in brief psychotic disorder, although these may differ somewhat in Europe and America. The symptom patterns include acute paranoid reactions and reactive confusion, excitation, and depression. Some data suggest that, in the United States, paranoia is often the predominant symptom in the disorder. In French psychiatry, bouffée délirante is similar to brief psychotic disorder.

DIFFERENTIAL DIAGNOSIS:

Clinicians must not assume that the correct diagnosis for a patient who is briefly psychotic is brief psychotic disorder, even when a clear precipitating psychosocial factor is identified. Such a factor may be merely coincidental. If psychotic symptoms are present longer than 1 month, the diagnoses of schizophreniform disorder, schizoaffective disorder, schizophrenia, mood disorders with psychotic features, delusional disorder, and psychotic disorder not otherwise specified must be entertained. If psychotic symptoms of sudden onset are present for less than 1 month in response to an obvious stressor, however, the diagnosis of brief psychotic disorder is strongly suggested. Other diagnoses to consider in the differential diagnosis include factitious disorder with predominantly psychological signs and symptoms, malingering, psychotic disorder caused by a general medical condition, and substance-induced psychotic disorder. In factitious disorder, symptoms are intentionally produced; in malingering, a specific goal is involved in appearing psychotic (e.g., to gain admission to the hospital); and when associated with a medical condition or drugs, the cause becomes apparent with proper medical or drug workups. If the patient admits to using illicit substances, the clinician can make the assessment of substance intoxication or substance withdrawal without the use of laboratory testing. Patients with epilepsy or delirium can also show

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psychotic symptoms that resemble those seen in brief psychotic disorder. Additional psychiatric disorders to be considered in the differential diagnosis include dissociative identity disorder and psychotic episodes associated with borderline and schizotypal personality disorders.

COURSE AND PROGNOSIS:

By definition, the course of brief psychotic disorder is less than 1 month. Nonetheless, the development of such a significant psychiatric disorder may signify a patient's mental vulnerability. Approximately half of patients who are first classified as having brief psychotic disorder later display chronic psychiatric syndromes such as schizophrenia and mood disorders. Patients with brief psychotic disorder, however, generally have good prognoses, and European studies have indicated that 50 to 80 percent of all patients have no further major psychiatric problems. The length of the acute and residual symptoms is often just a few days. Occasionally, depressive symptoms follow the resolution of the psychotic symptoms. Suicide is a concern during both the psychotic phase and the postpsychotic depressive phase. Several indicators have been associated with a good prognosis. See Table 7.5-1 for a summary of good prognostic signs in brief psychotic disorder.

TREATMENT:

- Hospitalization
- Pharmacotherapy
- Psychotherapy

6. CATATONIC DISORDER

Catatonia is a new diagnostic category in DSM-5 introduced because it can occur over a broad spectrum of mental disorders, most often in severe psychotic and mood disorders. It can also be caused by an underlying medical condition or induced by a substance.

Definition-

Catatonia is a clinical syndrome characterized by striking behavioral abnormalities that may include motoric immobility or excitement, profound negativism, or echolalia (mimicry of speech) or echopraxia (mimicry of movement). A diagnosis of catatonic disorder due to a general medical condition can be made if there is evidence that the condition is due to the physiological effects of a general medical condition. The diagnosis is not made if the catatonia is better explained by a primary mental disorder, such as schizophrenia or psychotic depression, or if catatonic symptoms occur exclusively within the course of delirium.

EPIDEMIOLOGY:

Catatonia is an uncommon condition mostly seen in advanced primary mood or psychotic illnesses. Among inpatients with catatonia, 25 to 50 percent are related to mood disorders (e.g., major depressive episode, recurrent, with catatonic features), and approximately 10 percent are

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associated with schizophrenia. The prevalence of catatonia due to medical conditions of substances is unknown.

ETIOLOGY:

Medical conditions that can cause catatonia include neurological disorders (e.g., nonconvulsive status epilepticus, and head trauma), infections (e.g., encephalitis), and metabolic disturbances (e.g., hepatic encephalopathy, hyponatremia, and hypercalcemia). Medications that can cause catatonia include corticosteroids, immunosuppressants, and antipsychotic (i.e., neuroleptic) agents. Catatonic symptoms may be seen in extreme forms of neuroleptic-induced parkinsonism or neuroleptic malignant syndrome, a rare, potentially life-threatening disorder associated with fever, autonomic instability, impaired consciousness, and rigidity.

DIAGNOSIS AND CLINICAL FEATURES:

DSM-5 criteria for the diagnosis of catatonic disorder due to a general medical condition (Table 7.5-3) include behavioral changes characteristic of catatonia, evidence of a physiological basis for the symptoms, and exclusion of primary mental disorders and delirium. The diagnosis of catatonia due to a mental disorder is used when the disorder occurs in a psychiatric rather than another medical condition (Table 7.5-4). In either case, the signs and symptoms of catatonia are similar; it is the etiology that differs. Behavioral changes may include motoric immobility or excessive activity, extreme negativism or mutism, peculiarities of voluntary movement, and echolalia or echopraxia. Waxy flexibility, a form of artificial posturing often evident on physical examination, may be present (Fig. 7.5-2). Lethal catatonia is a rare advanced stage of the disorder that features fever and autonomic instability and may be fatal.

DIFFERENTIAL DIAGNOSIS:

Differential diagnoses include hypoactive delirium, end-stage dementia, and akinetic mutism, as well as catatonia due to a primary psychiatric disorder. It is important to identify cases of catatonia occurring in the setting of neuroleptic malignant syndrome because the latter diagnosis can be fatal. Features suggesting neuroleptic malignant syndrome include autonomic instability and delirium in addition to elevated serum creatinine phosphokinase, white blood cell count, and serum transaminases.

COURSE AND TREATMENT:

Catatonia impairs a person’s ability to care for himself or herself and therefore requires hospitalization. In an excited state, the catatonic patient may represent a danger to others; hence, close supervision is needed. Fluid and nutrient intake must be maintained, often with intravenous lines or feeding tubes. The catatonic individual must be assisted with hygiene. The primary treatment modality is identifying and correcting the underlying medical or pharmacological cause. Offending substances must be removed or minimized. Benzodiazepines can provide temporary improvement in symptoms, and their use may improve patients’ ability to communicate and to care

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for themselves. ECT is appropriate for catatonia due to a general medical condition, especially if the catatonia is life threatening (e.g., inability to eat) or has developed into lethal (malignant) catatonia. The mechanism behind the efficacy of ECT is unknown.

7. PSYCHOTIC DISORDER NOT OTHERWISE SPECIFIED

Under the umbrella of psychosis not otherwise specified is a variety of clinical presentations that do not fit within current diagnostic rubrics. It includes psychotic symptomatology (i.e., delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior) about which there is inadequate information to make a specific diagnosis or about which there is contradictory information. It also includes disorders with psychotic symptoms that do not meet the criteria for any specific psychotic disorder, such as patients who present to hospital with persistent auditory hallucinations that are not accompanied by mood disturbances and that are not pathognomonic for schizophrenia.

Autoscopic Psychosis-

Although not included in DSM-5, autoscopic psychosis is of clinical interest. The characteristic symptom of autoscopic psychosis is a visual hallucination of all or part of the person's own body. The hallucinatory perception, which is called a phantom, is usually colorless and transparent, and because the phantom imitates the person's movements, it is perceived as though appearing in a mirror. The phantom tends to appear suddenly and without warning.

EPIDEMIOLOGY:

Autoscopy is a rare phenomenon. Some persons have an autoscopic experience only once or a few times; others have the experience more often. Although the data are limited, sex, age, heredity, and intelligence do not seem to be related to the occurrence of the syndrome.

ETIOLOGY:

The cause of the autoscopic phenomenon is unknown. A biological hypothesis is that abnormal, episodic activity in areas of the temporoparietal lobes is involved with the sense of self, perhaps combined with abnormal activity in parts of the visual cortex. Psychological theories have associated the syndrome with personalities characterized by imagination; visual sensitivity; and, possibly, narcissistic personality disorder traits. Such persons may likely experience autoscopic phenomena during periods of stress.

COURSE AND PROGNOSIS:

The classic descriptions of the phenomenon indicate that, in most cases, the syndrome is neither progressive nor incapacitating. Affected persons usually maintain some emotional distance from the phenomenon, an observation that suggests a specific neuroanatomical lesion. Rarely do the symptoms reflect the onset of schizophrenia or other psychotic disorders.

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TREATMENT:

Patients usually respond to antianxiety medication. In severe cases, antipsychotic medications may be needed.

Motility Psychosis-

Motility psychosis is not considered an “official” DSM-5 diagnosis but is of clinical significance. It is probably a variant of brief psychotic disorder. The two forms of motility psychosis are akinetic and hyperkinetic. The akinetic form of motility psychosis has a clinical presentation similar to that of catatonic stupor. In contrast to the catatonic type of schizophrenia, however, akinetic motility psychosis has a rapidly resolving and favorable course that does not lead to personality deterioration. In its hyperkinetic form, motility psychosis can resemble manic or catatonic excitement. As with the akinetic form, the hyperkinetic form usually has a rapidly resolving and favorable course. Patients may switch from the akinetic to hyperkinetic form rapidly and may represent a danger to others during the excited phase. Mood is extremely labile in these patients.

Postpartum Psychosis-

Postpartum psychosis (sometimes called puerperal psychosis) is an example of psychotic disorder not otherwise specified that occurs in women who have recently delivered a baby; the syndrome is most often characterized by the mother’s depression, delusions, and thoughts of harming either her infant or herself.

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